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A COMMENTARY
ON THE
DISEASES OF INDIA

BY

^c
NORMAN CHEVERS, C.I.E., M.D., F.R.C.S. ENG.

DEPUTY SURGEON-GENERAL H.M. INDIAN ARMY;

LATE SECRETARY TO THE MEDICAL DEPARTMENT, BENGAL; PRINCIPAL AND PROFESSOR OF MEDICINE

IN THE MEDICAL COLLEGE, AND FIRST PHYSICIAN OF THE COLLEGE HOSPITAL, CALCUTTA;

OFFICIATING INSPECTOR-GENERAL OF JAILS L.P.; FELLOW AND FORMERLY PRESIDENT

OF THE FACULTY OF MEDICINE OF THE UNIVERSITY OF CALCUTTA; LATE

PRESIDENT OF THE EPIDEMIOLOGICAL SOCIETY OF LONDON, AND OF

THE SANITARY DEPARTMENT OF THE NATIONAL ASSOCIATION

FOR THE PROMOTION OF SOCIAL SCIENCE



LONDON

J. & A. CHURCHILL

11 NEW BURLINGTON STREET

1886

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TO
ALEXANDER GRANT, Esq., F.R.C.S.

HONORARY SURGEON TO THE QUEEN

IN REMEMBRANCE OF AN UNBROKEN FRIENDSHIP OF THIRTY-FIVE YEARS

AND IN ADMIRATION OF HIS MASTERLY KNOWLEDGE

OF INDIAN DISEASE

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BY

THE AUTHOR



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PREFACE.

THIS Commentary is an outcome of a study of Indian disease maintained for thirty-eight years, upwards of twenty-four of which were mainly devoted to clinical observation in Bengal. The chapters, up to the end of that on the 'Diseases of the Respiratory System,' originally appeared in the 'Medical Times and Gazette,' between January 4, 1879, and December 19, 1885. This portion of the work has now been carefully revised, wholly recast, and, to a considerable extent, pruned and extended.

In publishing this volume I bring to an end a course of observation, thought, and study, which has occupied the whole of a professional lifetime.

My book on the 'Management of the Diseases of the Heart,' which appeared in Calcutta in 1851, terminated a series (*vide* list at head of the chapter on 'Diseases of the Circulatory System,' page 479) of contributions on Cardiac Pathology and Therapeutics commenced ten years previously. In a work on 'Removable and Mitigable Causes of Death,' published in 1852, I modelled into a system the scattered facts upon which our knowledge of English public hygiene then rested. In the early numbers of the 'Indian Annals of Medical Science' I published the results of an inquiry into the means of preserving the health of European soldiers in India. This was incomplete as regarded the health of armies in the field—a want which was supplied by surgeons of great military experience. Between 1855 and 1871, there appeared three editions of a work on 'Medical Jurisprudence in India.' I now offer some facts and opinions regarding medical and surgical disease in that country.

The alliance between the principal diseases of the two climates is so very intimate that I am fain to hope that these observations on Indian maladies will not be uninteresting to medical men in Europe.

I am much indebted to many friends, of both European and Indian experience, who have most kindly afforded me valid scientific aid in bringing out this Commentary—especially in giving, in reply to my inquiries, information regarding important subjects upon which they are (I grieve to say, of some good and eminent men, ‘were’) leading authorities.

One of my chief objects in producing this Commentary has been to represent the labours, in the vast fields of medical and surgical observation and practice which India affords, of my European and native brother officers. The necessity of not making this first edition of the book too voluminous has, however, prevented me from accomplishing this design to my own satisfaction; still, this volume may serve as a very imperfect, but considerable, *catalogue raisonné* of the invaluable contributions for which medical science is indebted to the noble body of physicians and surgeons in association with whom it was my great privilege to spend the years of my active official career.

32 TAVISTOCK ROAD, BAYSWATER :
August 1886.

In this volume I frequently cite the latest published statistics of disease, as given in the reports of the Sanitary Commissioner with the Government of India, in the four years 1880-83. To prevent unnecessary repetition, the strength of European soldiers, women and children, and of native troops and prisoners, in each of these four years, with the average strength of each class for the whole term, are given below :—

Strength of European Soldiers.

1880	.	.	.	.	.	.	.	59,717
1881	.	.	.	.	.	.	.	58,728
1882	.	.	.	.	.	.	.	57,269
1883	.	.	.	.	.	.	.	55,525
Average strength				.	.	.	.	57,809

Strength of European Women.

1880	.	.	.	.	.	.	.	4,184
1881	.	.	.	.	.	.	.	3,741
1882	.	.	.	.	.	.	.	3,539
1883	.	.	.	.	.	.	.	3,290
Average strength	.	.	.	.	.	.	.	3,676

Strength of Soldiers' Children.

1880	.	.	.	.	.	.	.	7,596
1881	.	.	.	.	.	.	.	6,548
1882	.	.	.	.	.	.	.	6,208
1883	.	.	.	.	.	.	.	6,015
Average strength	.	.	.	.	.	.	.	6,591

Strength of Native Soldiers.

1880	.	.	.	.	.	.	.	126,385
1881	.	.	.	.	.	.	.	114,612
1882	.	.	.	.	.	.	.	114,894
1883	.	.	.	.	.	.	.	114,830
Average strength								117,680

Strength of Prisoners.

1880	.	.	.	.	.	.	.	106,763
1881	.	.	.	.	.	.	.	100,841
1882	.	.	.	.	.	.	.	94,063
1883	.	.	.	.	.	.	.	88,174
Average strength								97,461

ABBREVIATIONS.

THE following abbreviations occur frequently in the notes :—

I. A.	stands for Indian Annals of Medical Science.
I. M. G.	„ Indian Medical Gazette.
C. M. T.	„ Transactions of the Medical and Physical Society of Calcutta.
M. T.	„ Medical Times and Gazette.
I. M. J.	„ Indian Journal of Medical and Physical Science.
S. C. R.	„ Annual Reports of the Sanitary Commissioner with the Government of India.

Errata et Corrigenda.

- Page 45, line 17, *for* western boundary *read* eastern.
„ 94, omit foot-note.
„ 98, line 17 from foot, *for* several *read* severe.
„ 114, line 12, *for* were *read* was.
„ 140, line 3 from foot, *for* 1881 *read* 1880.
„ 169, line 4, *for* fever malarial *read* malarial fever.
„ 175. In foot-note, *for* 68 *read* 170.
„ 202, line 6 from foot, *for* ti *read* it.
„ 210, line 2 from foot, *for* 121 *read* 122.
„ 262, lines 16 and 17, *the sentence commencing* 'Its most recently' *should follow the words* 'surgical proportions' in line 20.
„ 276, line 16, and 19 from foot, *for* 181 *read* 182.
„ 328, line 26, *for* Act *read* Acts.
„ 354, line 20, *for* 1346 *read* 1486.
„ 442, line 1 of foot-note, *for* Vol. 2 &c. *read* p. 167.
„ 714, line 23, *for* '2553 *read* 255'3.
„ 756, line 11 from foot, *for* case of *read* race of.
„ 789, line 8, *for* 260 *read* 60

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A
COMMENTARY
ON
THE DISEASES OF INDIA.

INTRODUCTORY REMARKS.

FIRST among the many new impressions which the mind of the observant physician receives, on landing in India, is a strong conviction that, before he can resume in his new home the practice of his profession with satisfaction to himself and advantage to his patients, he has a good deal to learn, and probably much more to unlearn. In this respect, the path of him who has previously gone through the admirable course of special instruction afforded at Netley is greatly facilitated; but many go to India who have not enjoyed the advantage of a Netley training; and I have long thought that, to them, some plain hints from one who has encountered the difficulties with which they will have to contend when they enter upon Indian practice will be useful and acceptable. I also think that this commentary may assist many young medical men at English and other European ports where many persons suffering from Indian diseases are constantly under treatment.

When I joined the Indian Medical Department, I had been somewhat unusually fortunate in having steadily worked in the hospitals and medical societies of London for twelve years. Still, the first thing that struck me, in practising in Bengal, was that doubt beset me on every side. As I advanced, I soon began to discover that my knowledge was more troublesome even than my ignorance; and that, while I had to learn how native patients ought to be dieted, when quinine can best be administered in remittents, and what are the uses of the chief drugs in the native materia medica, and to satisfy myself upon a hundred other questions of the greatest local importance, I found that I had before me the still more difficult task of divesting my mind of a host of strong opinions—such as that the medical man should never leave his house without his case of bleeding-lancets, that colchicum is ‘good’ for rheumatism, that mercury is supreme in hepatic diseases, and that cathartics are therapeutic agents of almost

Morisonian universality. In fact, I only avoided great mischief by the observance of strict moderation and the most vigilant caution.

My object is to give my readers, in the following commentary, that information of which I then found myself most in need.

Except for illustration, I shall avoid the discussion of points with which every well-educated young English medical man may be expected to be familiar: my wish being to enable my reader to bring his home knowledge up to the Indian standard.

Invaluable as most of the extant works on the Diseases of India are, they are all more or less fragmentary, no author having attempted to review the whole of the maladies, surgical as well as medical, which are prevalent in the East. In this commentary, I have endeavoured to give some notice of every disease in the observation of which the English medical man will find that he has something to learn on commencing practice in India, and also to set forth the leading characteristics in which the diseases of India differ from those of Europe.¹

A few remarks upon

GENERAL INDIAN DEATH-RATES

will fitly preface a review of the diseases to which mortality in the East is due.

In 1858 I showed that, from the commencement of the present century, the average annual rate of mortality in hospital, from all causes, among the men of H.M. and the H.E.I.C.'s European Forces, in the three Presidencies of India, had been 62·45 in the thousand.

Surgeon-General Hugh Macpherson found that, during the eight years ending 1853-54, the hospital death-rate of European troops in the Bengal Presidency was 63·38 in the thousand.

During these eight years, the total number of deaths out of hospital was 1,175—a large proportion, the occurrence of which was explained mainly by the fact that nearly the whole of this period was one of active and trying warfare.

The returns show that, during the last six years, the mortality rates of our *European army* throughout India, have been:—

1878	.	.	.	21·46	1881	.	.	.	16·86
*1879	.	.	.	24·28	1882	.	.	.	12·07
*1880	.	.	.	24·85	1883	.	.	.	10·88

In comparing these rates with those given as prevailing shortly before the great Mutiny of 1857, it is to be borne in mind that the

¹ See a report in reply to the inquiry *Des modifications que subissent certaines maladies, et en particulier les maladies infectieuses sous l'influence des climats tropicaux?* which I sent to the Congrès International de Médecins des Colonies, held at Amsterdam in 1883, p. 21 of the Report.

* Excluding troops serving in Afghanistan.

early rates belong to a period in which the health of the troops suffered greatly in war, and from all the destructive influences attendant upon field service in enemy's country. The hitherto unexampled lightness of the death-rate of 1882 and 1883, viz., 12·07 and 10·88, is in a large measure attributable to 'the remarkable abeyance of cholera among the European troops.' We can hardly expect to have many of such low death-rates within the next decennium. Still these exceptionally small Indian death-rates for two years—12·07 and 10·88 per mille—are seen to be comparatively large when we find it shown, in the Army Medical Department Report for the year 1881, that, during the ten years 1871–80, the death-rate of the whole British Army, at home and abroad, did not largely exceed them, being 12·59 per mille, while the rate in the West Indies was 11·02; in the United Kingdom 7·95; and in Canada 6·64.

The mortality of the *Native Army* of India is large. In the five years 1877–82, it ranged from 13·38 in 1877, to 37·79 and 41·12. In the last two years, in which there was much trying war service in Afghanistan, the death-rates in the regular native army of Bengal were so high as 53·61 and 53·15 per mille.

The death-rates in the *Jails* of India in the eight years, 1877–83, have been, per mille, 55·40; 31·88; 65·85; 77·90; 51·12; 44·42; 42·56; 34·28; the principal causes of death being dysentery, diarrhœa, and cholera.

The death-rates in the *General Population* of India, as obtained in 1882, under a still admittedly imperfect system of registration, ranged from 13·61 in Assam, to 29·99 in North-Western Provinces and Oudh. In Bengal the rate was 15·40; in the Punjab 27·03; in the Central Provinces 29·10; in the Madras Presidency 15·7, and in the Bombay Presidency 20·25.

Knowing the people and their modes of living as I do, I cannot but think that these rates are under-estimated, and that, as the system of registration becomes more accurate, the rates will be found to be considerably higher. Still it is to be borne in mind that these are mainly *rural* rates.

Dr. Beddoe¹ in an article on 'Mortality' gives the following *City* mortality rates for 1878: Calcutta, 37·7; Madras, 48·8; Bombay, 41·8. Nothing that parallels these rates has occurred to me in reading the recent mortality rates of the principal cities of the United Kingdom, one of the highest of which has been 29 per mille.

The death-rates of *European soldiers' Wives and Children* in India have always been deplorably high. Macpherson shows that, between the years 1838 and 1853, the mean death-rate of women in the three Presidencies was 35·47 per mille.

The Sanitary Commissioner gives the rates, between 1875 and 1883,

¹ Quain's Dictionary.

as ranging from 19·78 in 1882, to 29·20 in 1878. My friend Dr. Joseph Ewart, who has written¹ with great power on the 'Causes of the excessive mortality among the women and children of the European soldiers serving in India,' says that, through the kindness of Surgeon-General Marston, he has been placed in a position to give, side by side, the death-rates of the women belonging to the European troops serving in India, and in the United Kingdom; thus, one of the lowest Indian death-rates, that for 1882, being 19·78, the home rates for the four years 1877-80 were only 8·59, 8·65, 7·75, and 8·37.

The *Children's* death-rates, as given by the Sanitary Commissioner, are still more appallingly high. During the last nine years, they have ranged from 46·88 in 1883 to 79·73 in 1878. From the high authority cited above, Dr. Joseph Ewart shows that, in the four years 1877-80, the death-rates of soldiers' children in the United Kingdom were 24·08, 28·41, 26·86, and 30·28.

The outcome of this dreary array of figures is that—although, wherever we are able to draw an accurate statistical parallel, Indian death-rates are now generally lighter than they were in the evil old times of ill-built and over-crowded barracks and hospitals, battle, famine, and long campaigns, bleeding, calomel and ignorant midwives—English men, women, and children are at present subject to far higher death-rates in India than they are in any other large division of the British Empire.

The following questions deserve the deepest consideration from sanitarians and legislators.

Why should the last (up to 1882) decennial death-rate of our European soldiers in India—19·37—so vastly exceed that of their brethren in the West Indies, 11·02, even when it is admitted that the pestilential influences of the West Indies are less potent than those of the East?

Why should the mortality of soldiers' wives in India be at present, to use Dr. Joseph Ewart's words, 'about *three* times as heavy as in England'? We may well exclaim with him, 'the discrepancy is as enormous as it is appalling!'

Still again, why should the death-rate among soldiers' children in India, in the four years 1877-80, exceed, as Ewart has shown, the death-rate of barrack children in the United Kingdom by 26·25, 51·32, 50·20, and 30·28 per mille?

In placing these facts in the form of questions, I do not mean to leave it to be inferred that the causes of these terrible discrepancies are not to a considerable extent recognised; or that the Government and their medical officers are not devoting the most earnest interest and very great ability to the work of amelioration, which recent statistics show is generally advancing.

¹ Transactions of the Epidemiological Society of London, N.S., vol. ii. p. 47.

THE GENERAL CHARACTERS AND COMPARATIVE FREQUENCY
OF THE DISEASES WHICH PRESENT THEMSELVES TO
THE NOTICE OF THE PHYSICIAN IN INDIA.

On my first arrival in India, I was told by a medical man of great local experience, 'All the diseases of England occur in abundance here.' I, however, soon began to notice that this observation was not to be freely accepted. Several of the most prevalent diseases of England are either unknown or rare in India. Neither I nor any of my immediate professional associates ever saw a case of scarlatina in Lower Bengal, although the disease has been carried to Bombay and Madras in troop-ships. I never treated a case of typhus in India. The question, does typhus occur in India? is open to debate. True enteric fever prevails from time to time in Calcutta, and it appears to be increasing in frequency; but it is happily not common. This fever probably occurred in India in old times; but we have no evidence of its considerable prevalence there until the Calcutta outbreak of 1858 was observed by Edward Goodeve. Scarcely any Indian station for European troops is now exempt from its visitations. True scirrhus is very rare, both in natives and Europeans. A good many cases of medullary cancer occur among natives of the poorer classes. A few years ago, erysipelas, both in its idiopathic and traumatic forms, was almost absent from Calcutta. I never treated but one case of idiopathic facial erysipelas in India. But, of late years, the traumatic form has become nearly as prevalent in all the hospitals of Calcutta as it is in those of London. The idiopathic disease seems to be upon the increase, and with it has appeared its congener, puerperal fever, previously a rare disease. Those conditions of the glands and joints which are commonly known as 'strumous' occasionally present themselves in poor natives, but they are by no means prevalent in the plains of India; they are commoner in the hills. I should have known very little of this class of diseases had I depended solely upon what I saw of it in Bengal.

The physician will not have practised long in India before he meets with a case of phthisis; but the disease is comparatively rare in that country. Thus, unless a European takes latent tubercles with him, he can scarcely become the subject of tuberculosis in India, except under very exceptional circumstances. Natives suffer from phthisis—but, as will be shown in my chapter on this disease, very much less frequently than the inhabitants of the British Isles do. It has been said that, at home, the Englishman generally dies at the lungs; it is equally true of the native of India that his natural death is by some form of intestinal flux. The Anglo-Indian is not at all peculiarly

liable to either pulmonary or intestinal disease; and, in retirement to his native land, he has, I think, a decided advantage over his contemporaries who have remained at home, in the fact that, while he is not remarkably subject to bronchitis, his bronchial tubes have been far seldomer visited by catarrh than theirs have, and have therefore a fair chance of retaining their integrity longer. Among the ill-fed and badly clothed poor of India, chest inflammations occur during the cold season and rains, which try them very severely; but cases of pneumonia and pleurisy are far less frequent in Bengal than in England. Among the poorest natives, pneumonia of the apex is rather common. Destructive epidemics of pneumonia, generally complicated with pleurisy, are of rather frequent occurrence in Upper India. Apart from hepatic abscess and kirrrosis, diseases of the liver are not prevalent in India. Sallow complexions and jaundiced eyes are much more frequently seen in London than they are in Calcutta. Those forms of dyspepsia which so frequently render life almost unendurable in England are nearly unknown in India. There, if food disagrees, it may cause diarrhœa; but heartburn, gastric distress after meals, and dyspeptic hypochondriasis, very rarely occur. This is, I believe, due to the fact that, in England, the skin generally acts imperfectly for nine months in the year, and the consequence is chronic portal and hepatic and gastric congestion. India, too, has its portal congestions, but they are not generally chronic, the skin for nine months in the year doing its duty fully.

Measle, *Cysticercus Tenia mediocanellata*, has been demonstrated by Aitken as common in ration beef throughout the Punjaub, &c., but I believe that it has yet to be shown that hydatid disease, *Echinococcus hominis*, ever originates in any part of India. Tape-worm is not uncommon among the lower class of Europeans. I think that all of my many patients acknowledged that they were eaters of raw meat. European ladies and children occasionally suffer from lumbrici. Native children are very liable, and native women of the poorer class are so almost invariably the victims of these parasites that my house-physician was instructed to order infusion of quassia as the vehicle of every fluid medicine prescribed in the female ward.

True acute rheumatism occurs distinctly, but very rarely, in Lower Bengal. I seldom met with more than one or two cases every year in Calcutta. In these, cardiac complication was liable to occur. Two of the cardiac cases proved fatal within my observation, one in a native lad in my own ward, the other in a European gentleman whom I visited with a friend. Chronic cardiac disease, of rheumatic origin, is very rarely produced in Bengal Proper. Gout is extremely rare in India. I once attended an English civilian suffering from moderate symptoms of this malady. Old Indians, retired in England, who have been exempt from this disease in the East, are liable to attacks. Sub-

acute arthritic and muscular rheumatism is not uncommon, but is usually mild and transient. The so-called chronic 'rheumatic' affections of natives will generally be found to be of malarious origin, and may, therefore, be more properly termed 'neuralgic' and be treated as such.

True morbus Brightii is certainly far less common in Bengal than it is in London, still cases frequently occur in natives, especially the intemperate; and I can recall several fatal cases in officers who have lived carefully. As frequent attacks of intermittent fever induce cirrhosis of the liver, malarious influence—and probably also excessive sweating—appears to be capable of setting up organic renal mischief. I have seen a good deal of simple albuminuria, both in young and old Indian officers, apparently unassociated with permanent disease of the kidneys. Such cases, well sifted, would probably range themselves under the head of intermittent albuminuria dependent upon spanæmia and serous hæmorrhage.

Croup is said to occur in the hills of India, but I think that this statement requires confirmation. I never saw or heard of it in Bengal, where, however, laryngeal diphtheria is not infrequent, and spurious croup is very prevalent during the rains.

It will thus be seen that, although the popular idea at home is that most of the various climates of India are very deadly, and although a military surgeon has recently (not without reason) called India 'the worst country in the world,' those Europeans who seek fortune in the East may fairly reckon upon a very singular immunity from some of the gravest diseases of Europe; while, as regards the deadliest maladies of the East, well-to-do Europeans are very little liable to cholera, the premonitory diarrhoea of which may generally be checked. Whereas attacks of fever, dysentery, and hepatic abscess are almost always avertible when quinine and ipecacuanha are promptly and judiciously employed. Parturition is, doubtless, more generally easy (except among immature native girls) in India than in Europe; but the tendency to post-partum hæmorrhage, owing to spanæmia and deficient muscular tone, is certainly greater.

Except the nobility and landed gentry of England, no class in the world are more 'well-to-do' than European officers and gentlemen in India, or generally lead more temperate, regular, and moral lives. In old times, India gained most of its reputation as a deadly climate from the writings of ships' doctors, who observed disease as it occurred in sailors, debilitated by sea-scurvy, on the loose in Indian ports, at a time when mercury and bleeding were the leading therapeutic indications. Now mercury, leeches, and bleeding-lancets are almost unknown in India; and the consequence is that, although occasionally attacked with malarious fever and dysentery, a large proportion of educated English gentlemen and ladies and well-conducted soldiers

live very healthy lives in India, and retire with fairly preserved constitutions, which, with due care, bear the change to England not only with impunity, but with singular advantage.

Still, almost universal as temperance is among well-educated Europeans in India, it was probably the exception in all classes until of late; and even now, European roughs and men who cannot devote their leisure either to books or to field sports, are generally drunkards. Now, as of old, many an English mother exclaims, 'India killed my poor boy!' where, could she only know the truth, she would perceive that his mode of living was so intemperate and so grossly imprudent as to be incompatible with longevity in any climate.

On the darker side of the picture, we have to observe the fact strongly defined by one of my professional friends in the axiom, 'We, in India, work remarkably well, but we are nearly all a parcel of screws.' That is to say, the European constitution deteriorates in India, as it does everywhere else, as life advances, but probably by different modes of decay and mal-nutrition. Thus the old Indian is generally either thinner or much fatter (both externally and internally) than he ought to be; and there must be something very wrong, constitutionally, with those multitudes of Europeans in India who cannot swallow a dose of Epsom salts or of seidlitz-powder without imminent danger of Asiatic cholera,¹ or stand for five minutes in a draught without shivering and falling into a paroxysm of intermittent fever. It is not so much by violent attacks of disease that the constitutions of prudent Europeans in India become impaired, as by the insensible but constant operation of mischievous climatic influences, such as the marsh-poison and the violent transitions of temperature, producing undue cutaneous action alternating with portal and other venous congestion, from which no one can entirely escape. True, we drain our cities and cantonments, spend a large proportion of our income in obtaining healthy residences, and clothe ourselves all the year round in outer garments of flannel, knowing that our predecessors often got their death by dressing in linen. Still, these morbid influences are constantly at work, producing fatty, fibrotic, and amyloid changes in the viscera, and deteriorating the blood and loading it with waste material which the degenerating liver, kidneys, and other organs can no longer adequately eliminate. We, the Indian doctors of the old school, have, I think, done our part in investigating and contending with the outward maladies—fevers and fluxes—which abound in India; but our successors, who have learnt from us to regard pathology as physiology gone astray, may hope to effect very much more than we have achieved in the prevention and treatment of the constitutional diseases of India.

¹ I never gave a dose of either of these medicines in India, and I never gave more than half the ordinary dose of any purgative at a time to a European, recommending the patient to repeat the dose if needful. The second dose was scarcely ever required.

INFLUENCE OF TEMPERATURE UPON DISEASE IN INDIA.¹

The subjoined table² will afford a fair idea of the prevailing temperature in the Plains and Hills of India :—

	Calcutta	Rajshahye	Delhi	Rawul Pindce	Simla	Madras	Bombay
	degrees	degrees	degrees	degrees	degrees	degrees	degrees
January . .	63·4	68	56	49·8	40·0	79·65	Mean temperature of year, 81
February . .	74·2	75	61	57·1	44·1	80·64	
March . . .	82·9	87	72	55·1	53·4	83·24	
April . . .	86·6	89	83	74·2	61·3	85·75	
May . . .	89·0	93	91	86·9	66·3	86·80	
June . . .	86·2	88	92	90·9	80·9	87·67	
July . . .	84·1	87	86	85·5	75·5	86·00	
August . .	82·6	88	83	83·2	78·1	86·00	
September .	83·8	89	83	85·7	70·0	84·8	
October . .	81·1	84	77	76·2	67·9	84·60	
November .	75·4	77	65	45·0	52·3	78·51	
December .	66·9	—	58	—	46·1	79·31	
	inches	inches	inches	inches	inches	inches	inches
Total rainfall of the year	62·0	34·61	20	—	70	48	80

Effects of Indian Cold in the Production of Disease.—A stranger, looking at this table, will probably remark, ‘Why, cold weather is never experienced in the plains of India, however much it may prevail during a winter in the hills!’ To this we must reply, whatever the thermometric readings may be, cold and its ill effects are by no means unknown to the inhabitants of the plains. The ‘cold season’ in Ceylon, Madras, and Bombay certainly little deserves that name. During a morning’s open-air work in Bombay, on November 30, some years ago, I said to myself, ‘I am in danger from this dreadful heat!’ During the railway journey up the Ghauts, that afternoon, the heat was pleasant. Soon after nightfall, we reached the summit of the ascent, and there, all unprepared, we rushed into a wind so piercingly chilly that it gave me an attack of my Chittagong fever. The cold season in Calcutta is, to people in sound health, very pleasant and invigorating; but to many it is, at least at its commencement, only a wretched ordeal of chill and congestion.

During my first and second winters in India, I was tolerably healthy

¹ By those who desire to study the climatology of India, admirable guidance will be found at p. 382 of Sir Joseph Fayrer’s *Tropical Diseases*, 1881, and in his first Croonian Lecture, 1882.

² This table is compiled from data given by Sir Ranald Martin, Dr. D. Stewart, Dr. John Macpherson, Mr. Hugh Macpherson, Mr. Joseph Bedford, Mr. Alexander Grant, the *Encyclopædia Britannica*, the *Madras Topographical and Statistical Reports*, and Chevers’ ‘Health of European Troops in India.’

and thought the cold weather most enjoyable. After that I became subject to tertian fever and its congener neurotic asthma. I then began to dread the cold, and to understand the expression of a Highlander in India, which is, I think, cited by Sir R. Martin—‘I can rejoice in the rude blasts of Caledonia, but this damp cold is terrible!’ Thenceforward, for four-and-twenty seasons, I scarcely ever escaped an illness, of from three to six weeks’ duration, at the beginning of the cold weather, except in two winters spent in England. The expression, ‘This is more like cold water than cold air!’ constantly suggests itself to the invalid in Bengal. The native poor, who are insufficiently clad, and can ill afford to buy fire-wood, suffer miserably from cold, and their dwellings are constructed with a view to resist cold rather than heat. And yet a black frost is impossible in Calcutta; a little hoar-frost is sometimes visible. Northward, the cold increases in severity up to Peshawur, and the duration of the winter increases from about three months to five. Stoves are not always provided in Calcutta houses; but, during the cold season, a greatcoat and a rug are indispensable during the early morning and evening drive. Still, except for a few days about New Year’s Day, the sun is so powerful as to prevent Europeans from walking about with comfort in the middle of the day. The Indian cold season is to be feared because it rather suddenly checks the cutaneous action of those who have been in a state of profuse perspiration for the preceding nine months, and because, it being the period of greatest drought, paludal poison is up and doing. What one of my colleagues terms the ‘masked malignity of the Bengal winter’ displays itself in most of the evils which result from arrested cutaneous action and visceral congestion in many forms—dysentery, acute suppurative hepatitis, congestive malarious remittent of the cold season, bronchitis, hæmoptysis, and intercurrent pneumonia in phthisis, hæmatemesis in kirrrosis of the liver, morbus Brightii, catarrhal affections, chronic bronchitis in aged natives, pneumonia, &c. As a general rule, every European in India looks forward with pleasure to the cold season. It is a time of amusement, and its sensations and aspect recall the idea of home. It gives new vigour to the healthy, and the delicate generally find themselves stronger before it is at an end. It is certainly better to fall ill before than after this season, because, in the former case, the cold weather generally aids the European’s recuperative power, whereas, in the latter, the invalid has to undergo the ordeal of nine months’ exhausting heat.

Effects of Indian Heat in the Production of Disease.—The ordinary action of Indian heat upon Europeans, is, I think, generally misunderstood at home. When he returns to England, the Anglo-Indian is generally pale and averse to long walks: but the fault in his complexion is generally caused by living in a darkened room,

although it may, in sickly persons, be due to anæmia; and his apparent inactivity mainly arises from the fact that people in India do not go about their work a-foot. He therefore usually regains colour and muscular activity almost as soon as the blisters on his feet are healed. It is also often imagined that the European in India is languid and debilitated. On the contrary, I believe that more cheerful work is done there than here. In India, there is none of that dull sense of cold which depresses the spirits and renders the mind sluggish: the hot clear air of India moderately excites and exhilarates. The Indian official rises at five, works cheerfully in office from ten till six, and then too often goes to a dinner-party, which, although it is a strictly temperate affair, is a mistake, because it robs him of that evening quiet and rest which he requires, and does not allow him to go to bed till twelve. Thus many work pleasantly for years, never sleeping in the day-time. Free action of the skin, although an essential to health in hot climates, is generally troublesome in Lower Bengal, in consequence of the great humidity of the atmosphere; and in some it is excessive, being at once a result and a cause of debility. On a very hot day in England, the idea that a cold bath would be pleasant at once suggests itself. I never exactly felt this in Lower Bengal. One feels wet rather than hot, in consequence of the slowness of evaporation. So great is the moisture of the climate that one who lives quietly suffers but little from thirst in Bengal. It is the reverse in the dry up-country heat. Hence, we used to notice that, when a man from the North-Western Provinces came to an appointment in Calcutta, he always began with a large *surai* of drinking-water on his writing-table. This generally disappeared in a few days. One of the very worst consequences of Indian heat is disturbed sleep. There are years in which the dweller in Bengal Proper may declare that he has not enjoyed perfect sleep for nine months. It is especially in this that the Indian cold season, or a cool 'rains,' or a sojourn, however short, in England, proves so validly invigorating to the Anglo-Indian constitution. If there is any one point in which, physically, the denizen of England has especially an advantage over the dweller in Bengal, it is in this: he sleeps—not longer, or oftener, or more heavily—but sounder and more refreshingly.

There is a great deal of heat-stroke (no longer erroneously termed heat-apoplexy) in India. The Hindu and Mahomedan natives, who are only naturalised foreigners, not the aboriginal inhabitants of India, also occasionally suffer from heat-stroke, as happened when Suraj-ud-Dowlut suddenly moved his army down from Moorshedabad upon Calcutta, in heat-stroke weather, immediately before the setting in of the rains. Great outbreaks of heat-stroke, such as occurred in England in the summer of 1878 and in New York eighteen years ago, are less frequent (although they do occasionally happen, as in the Mutiny

year) in India than in Europe ; because, in the former country, the danger is always apprehended and guarded against. I have long looked upon heat-stroke, as we see it in India, as being more generally due to some error in the constitution than as being the sole work of the heat, which has, in many cases, merely acted as an exciting cause. The noxious exhausting effect of excessive solar heat upon the nervous system of man is by no means certain and uniform. Thus, if a hundred men fall into boiling water or enter an atmosphere of carbonic oxide, all, without a single exception, will of course be scalded or choked ; but if, as occasionally happened in old times, a company of European soldiers, having to take the short march from Dum-Dum into Fort William, started an hour later in the morning than they ought to have done, all would arrive much knocked up ; but probably only some five or six would be brought into the main guard with heat-stroke. If we could, looking at these sufferers, judge (as I do not pretend that I could do immediately) what the diseased state of each precisely is, we should probably read their conditions somewhat as follows : — This rather corpulent sergeant is beginning to grow old ; he has served long in India, and has lived intemperately ; he has some kirrrosis of the liver and organically defective kidneys ; he is suffering from cholæmia and uræmia, rendered dangerous by heat-stroke. This muscular young man is epileptic ; he fell in a state of eclampsia. This well-conditioned soldier started feeling rather chilly and unwell ; the sun's influence struck him down just as the hot fit of intermittent fever came on. This man's small intestines will, on post-mortem examination, be found to contain cholera stools. Who is this poor blue-jacket among the red ones ? He is a European sailor on the loose, who reeled in with the party. He has drunk a quantity of liquor which he might just have been able to carry in cold weather, but he was so nearly upon the verge of dangerous alcoholic poisoning that his brain could not endure the additional trial of exposure in heat-stroke weather ; so that here we have a very common case of what is called insolation in Calcutta. This immature lad is a recruit of delicate frame, scarcely equal to the weight of his accoutrements. When he became greatly fatigued on the march, extreme heat completely overwhelmed the well-nigh exhausted power of his nervous system ; and thus we have, in his person, the only case of true and uncomplicated *ictus solis* in the party.

Although most prudent Europeans avoid, as much as possible, direct exposure in Indian heat-stroke weather, it takes a great deal of such exposure to produce this kind of heat-shock in a perfectly sound nervous system. There must, apparently, be some weak point at which the enemy can enter. An elderly Highlander, of herculean proportions, who had generally enjoyed robust health, told me that, during his long career as a shipbuilder near Calcutta, he had daily

occupied himself in his dockyard almost without regard to the sun. On one occasion, however, he had attempted only to round the corner of a building, coming out at one door to enter at another, when the sun struck him down. Doubtless there was, at that moment, some 'screw loose' in his constitution, although he was not aware of it. Upon disease generally, excessive heat has a very injurious effect, rapidly exhausting the phthisical and the subjects of large open hepatic abscesses by hectic and colliquative sweating, and especially searching out any weak point in the nervous system. Thus he who is epileptic in India frequently recovers in a temperate climate. I used frequently to tell my students, when the rains set in, that it was nearly certain that all our brains and spinal cords were fairly sound, or we should not have passed through the hot weather so safely as we had done. In ordinary Indian weather, I very rarely lost a case of delirium tremens; but I have frequently said to my house-physician, when admitting a typical case on a heat-stroke day, 'He can scarcely outlive the day'; and this because experience had taught me that a temperature which healthy brains could barely endure must almost certainly overwhelm and arrest the action of this exceedingly embarrassed one. The exhausting effect of heat is almost as operative in the night as by day. Thus, on my morning visit, at this season, I not unfrequently missed a case which I had expected to last some days or weeks longer. He had failed during the great still heat of the night. On the afternoon of a heat-stroke day, I was called by a brother officer to see a young English lady just confined with her first child. There had been nothing unusual in the case, and the loss of blood had been perfectly normal. Still she sank and went out in our presence. She did not appear at all unhealthy, but it was said that she was less strong than her sisters. I felt convinced that, had she been confined in moderate weather, she would have lived. In nearly like manner, another young lady sank, about three weeks after her first confinement. The friends all attributed her death to prickly heat (*lichen tropicus*). Both of these patients failed to obtain, in heat-stroke weather, that recuperative nervous energy which the trials of a first parturition demanded in them. Ten summers ago, I was so unfortunate as to be at Hastings in heat-stroke weather. My landlady informed me that my bedroom was usually occupied by a consumptive lady. 'A few nights there,' I said, 'would inevitably kill her.' 'Yes,' was the reply, 'in very hot weather she goes to Tunbridge Wells.' I believe that all that we have here considered, with regard to excessive heat in India, applies largely to the condition of our sick in the hot weather of temperate climates.

Of all the vicissitudes to which the climate of India is liable, none interfere with health so seriously as the *rapid transitions of its temperature do*. Steel can alone bear instantaneous change from heat to

cold, and steel is generally wanting in Indian constitutions. Hence the invariable prevalence of sickness, especially from dysentery, hepatitis, and other abdominal diseases, at the commencement of the cold season and rains. The rainy season has always held a very evil reputation, even with medical men. The result of my own observation is that, when the rains set in heavily, continue evenly, and cease late, this season has not, in itself, many of the elements of unhealthiness, but it is constantly happening that he whose constitution the previous hot season has thoroughly exhausted, becomes the victim of serious congestion when the rains set in. The abrupt change in temperature is, in fact, *too good* for him. If he could have been placed, as suddenly, in England on St. Swithin's Day, the violent transition would scarcely have hurt him less.

In India, as a general rule, people bathe every day and perspire freely. I recollect to have been told by Dr. Daniell, who made some very important observations on the Western Coast of Africa in its medical aspects, that, upon first shaking hands, a native chief told him that he had a good prospect of escaping the diseases of that country—his hand was moist. In India, as elsewhere, excessive perspiration is, of course, a sign of debility; but to the healthy freedom with which his skin acts, the European in India is mainly indebted for his power of enduring great heat and cold with impunity, and for his singular immunity from certain forms of disease, such as gout and rheumatic fever, and the most distressing forms of dyspepsia. With a sound brain and perfectly acting organs of excretion, the European has an equal prospect of health during the time of his service in India, and after his return to England. In a man thus happily constituted, the balance of action between the several excreting organs is maintained with admirable equipoise; the skin acting freely, there is no portal or thoracic congestion, and hence no constipation, 'biliousness,' or catarrh. Under such conditions of health, the organs whose action is reciprocally compensating act for one another with the greatest ease. Thus, when, in the hot season, a healthy European leaves the Plains for one of those Hill Stations where the ascent is sudden, a rise of a few hundred feet accelerates the heart's action, checks perspiration, and causes so much diuresis, that he is apt to become alarmed and to consult the doctor when he reaches the station.¹ In a day or two, he probably gets a sharp attack of diarrhœa known as the 'Simla trots'—evidence that the liver and intestinal mucous membrane are acting vicariously for the skin. The same perfectly physiological tendency to compensatory diuresis, with some liability to diarrhœa, generally annoys the Anglo-Indian for a few months after he returns to Europe. This leads us to perceive why the climates of the Hills and of Northern

¹ The first notice of this fact was, I think, by Dr. Percival Lord. Vol. i. of the *Trans. of the Med. and Phys. Soc. of Bombay* for 1838, p. 280.

Europe are so uncongenial to the Englishman who is the subject of confirmed organic disease, and why, as we have shown, the setting in (1) of the rains and (2) of the cold season, always refreshing and salutary to those in sound health, is so much to be dreaded by those whose innervation has become exhausted by the preceding hot weather, and by those whose constitutions are seriously impaired from malarious influence or any other cause.

Few hygienic measures have tended more validly to secure the health of Europeans in India than the abandonment of the dangerous luxury of linen garments and bedding, and the introduction of calico and of light flannel outer garments. Nothing can more refresh the healthy European than one of the thunderstorms which generally occur in Calcutta on or about the Queen's birthday in May. But, to profit by this sudden change, he should by day have an extra waistcoat, and by night a rug, always ready. He who is so incautious as to get a thorough wetting, or, as is often the case, to struggle with the storm in shutting his venetians blown open in his first uneasy sleep during the almost unendurably sultry calm of atmospheric tension which preceded the storm, will be exceptionally fortunate if he escapes that congestion of which the outward expression is dysentery, fever, or cholera. To an influence of this kind much of the acute hepatic disease, tending to abscess, for which the otherwise pleasant station of Bangalore, in the Madras Presidency, is or was notorious, was attributed. The day and night temperature up to midnight, in the hot weather, is very high. Towards dawn, a cold wind comes down from the hills, and, sweeping through the barracks, chills the over-heated and perspiring sleepers, and frequently sets up serious visceral congestion.¹

A few examples, out of thousands, of the ill effects of sudden transitions of temperature in India will suffice.

A gentleman came to me, not long before I left Calcutta, with facial paralysis. Before he could speak I inquired, 'On what night did you last sleep in your verandah?' He replied, surprised, 'How do you know that I sleep in my verandah?' 'Ah!' was the answer, 'doctors know many more remarkable things than that.'

A celebrated general, who was said to be the largest man in the Queen's Army, was taking a stroll near one of the Hill Stations. He felt hot, and seeing a pretty stream falling over a rock, he undressed and stood under it. Suddenly, he felt what he described as the sensa-

¹ Dr. A. Nairne has described (*Brit. Med. Jour.* February 1, 1879, p. 150) what he considered to be a new Indian disease, which prevailed extensively among the native coolies at South Sylhet. He believed (although his paper is wanting in the data for such a conclusion) that one of its leading characters was renal catarrh. He attributed it to working almost naked in unusually cold and windy weather at the breaking up of the rains.

tion of a bayonet-thrust through his liver, and fell in a heap. An attack of acute hepatitis immediately followed.

An officer, feeling the night heat extremely, got into a cold bath; tetanic spasms at once set in. Bishop Heber died in a bath immediately on his arrival at Trichinopoly after a fatiguing journey in a palankeen. I have seen a violent attack of colic excited by taking a cold bath in India.

One who was a dear fellow-student and brother officer of my own had an attack of acute dysentery in the rains. He recovered with great difficulty, evidently remaining weak and out of health. Early in October, the monsoon broke up in a violent night storm; symptoms of active dysentery had set in when I was called to him in the early morning, and he died in nine days from gangrenous colitis.

The master of the Calcutta Sailors' Home, a singularly fine middle-aged man, went to bed well one very hot night in the cholera season. When perspiring greatly, he was called up to pursue a thief, going in the thin light cotton drawers which all wear at night. Immediately upon his return, he was attacked with cholera, and died in a few hours. His trousers would have saved him.

In Calcutta, whenever a heavy storm occurs during the cholera season, the violence of the endemic is certain to abate—with this important reservation, that, when the storm occurs at night, several patients (chiefly Ooriah palkee-bearers who are ill-lodged and ill-clad), whom it has chilled, are generally brought in with cholera the next morning.

Again, however hot the weather in Calcutta may be, that marked fall of temperature which usually takes place between midnight and dawn generally affords a few hours' refreshing sleep. This cold of the 'small hours,' so beneficial to the healthy, was an influence which I always dreaded extremely, and endeavoured to guard against most vigilantly in practice. It is at this, the most critical period of the twenty-four hours, that phthisical patients are commonly attacked with hæmoptysis. It is then that anæmiated women and ill-fed native men, suffering from any form of exhausting disease, become suddenly collapsed, and that delicate children are attacked with diarrhœa or spurious croup. It is then that the onset of local congestion may be anticipated in the subjects of variola, measles, and remittent fever. This is usually the time at which premonitory diarrhœa gives place to true cholera; and now it is that the moribund native sick, with whom my wards were always crowded, become the subjects of what is perhaps best described as hypostatic congestion, which, causing serous oozing into the cerebral and thoracic cavities—especially into the pulmonary air-cells—leaves the victim comatose and suffocating at about the hour of our morning visit.

Here the sequence of depressant causes, eventuating in a tendency

to death, is perfectly clear—exhausting and painful disease ; eighteen hours of extreme heat and almost absolute sleeplessness ; the neglect of tired nurses in the administration of nourishment ; a sudden and considerable fall in the temperature of the air ; chill, congestion, effusion.

My hospital was certainly the most draughty receptacle for the sick that it has ever been my ill-fortune to attend ; consequently, these evils prevailed there very conspicuously ; but the principle at issue applies equally to the generality of sick-rooms, not only in India, but in Europe. Against this danger, my assistants and nurses were instructed to be constantly prepared. Sinapisms, cordials, and hot concentrated beef-tea, shawls, extra blankets, and hot-water bottles, were to be always ready. The change was to be clearly anticipated, and additional aid was to be promptly summoned. In this way, the period of greatest danger may frequently be tided over. These remarks will be found applicable to most of the grave diseases which we are about to consider. The generality of observers have recognised the above facts ; but they are not always sufficiently borne in mind in practice.

That venerable master of sanitation, Edwin Chadwick, informs me that he has lately communicated to the Indian Government his plan, brought forward at the Epidemiological Society in 1885, of ventilation with air from superior layers in place of inferior layers, by which he anticipates that, during the prevalence of great heat, we should obtain for apartments pure cool air free from contamination by disease-germs.

INDIAN DIETETICS.

Food of Europeans.—Although the provision made by that admirable service, the Indian Commissariat, for rationing our armies in time of war is proverbially excellent, our men have, on several occasions, suffered much from want of proper food when campaigning. During our early Burmese wars, nearly all disease was complicated with scorbutus ; and scurvy was almost universally prevalent among the heroic defenders of Lucknow. My friend, Sir Joseph Fayrer, tells me that so extreme was the want of all garden vegetables—except a very scanty supply of the English kinds in tins and bottles—that some of our men used to brave the never-ceasing fire of the enemy to gather *sag* (a wild spinach which sprang up scantily among the *débris* of their shattered defences) that they might sell it, weight for weight against silver, for the starving ladies and children. Our activity in annexing and occupying distant territory has frequently been so great as to outrun our means of providing sufficient food for the new occupants. This was the case in Scinde, of which I shall have to speak as a district notorious for scurvy. For a long time after we formed stations

in Lower Burmah, the want of adequate supplies of fresh meat, and the almost entire absence of fruits and vegetables, were very severely felt. This will now have to be looked for in our occupation of Upper Burmah. When I went through the province of Assam, in 1866, there was no baker's bread at any of the stations above Tezapore; rice and biscuit, brought up from Calcutta in the steamers, supplied its place whenever I was entertained by my brother officers. When I reached the large station of Dibrughur, the report that there would be beef for dinner that day was causing unusual excitement among the European residents.

These are, however, very exceptional instances as regards India generally, where the supply of food is so good and so varied that excess rather than want is our danger. The European in India is most fortunate in his cooks, and the want of such service is one of his severest trials in his retirement at home. The philosopher who defined man as 'a cooking animal' certainly had not in view our otherwise excellent sisters the women of England! In India, every native man and woman is a 'good plain cook,' and every *bowurchee*, however unskilled, sends daily to his master's table such a dinner as places in great danger of becoming a gourmand the ex-student who has for years grown more and more dyspeptic in battenning upon the smoked and scorched flesh with which his lodging-house drudge has provided him. Still, unless the Indian master be constantly upon his guard, his table servants will lose no opportunity of placing before him the very bad meat and fish with which India abounds. The ancient sages, who made the eating of grass-fed beef and measly pork a breach of religion to the Hindoos and Mohammedans, were clearly masters in sanitary science. Such beef as this is best judged by the physician, who attempts in vain to obtain good beef-tea out of its fibres. I recollect, with disgust, a splendid mess-table, for the support of which the officers paid 250*l.* a month, groaning with viands not one of which a man experienced in Indian food would dare to eat. No fresh pork, except that which has been fed by reliable Europeans, ever appears on a gentleman's table in India; but the poor Portuguese half-castes eat a great deal of diseased bazaar pork, and our European soldiers used to consume it, to the manifest detriment of their health. Except for a short time at the very hottest season, when fowls, oxen, and sheep fall out of condition, and when the best food is fish, our servants, in the large cities, can always give us good chickens and pigeons, and well-fed beef and mutton, if we absolutely insist upon having them.¹

¹ At out-stations, where the European residents do not maintain a 'mutton-club' by subscription, such notices as this, which I give *verbatim*, frequently appear: 'Raj-ub-Ali, butcher, presents his compliments to the Nobility and Gentry of Chittagong. On Monday next he will kill a fine *gram*-[bean] fed ox, with plenty of lean on his beef.'

Anyone who imagines that Indian heat spoils our appetites is greatly mistaken ; and many who, in England, have been accustomed to dine off a plain joint and pudding, and to take little beyond an egg or a rasher with their bread-and-butter at breakfast, often fall into the great error of eating highly-seasoned meats at each of their three daily meals. I recollect saying to a healthy-looking acquaintance, ' How is it possible to eat a heavy tiffin and a full dinner ? No man can eat two dinners a day.' His reply was, ' I have done so for a good many years.' He soon afterwards had to leave with liver disease. A neighbour of his, who lived very similarly, had a liver which reached nearly to his pelvis. I have seen almost as much disease among mess-managers, who are generally admirable judges of food, as among canteen sergeants, who are unfortunate in having constant access to alcoholic drinks.

I do not at all mean to say that gluttony is common among Europeans in India, but the free provision of rich meats at each meal is a source of danger to all, and proves a snare to some. For many years, my only meals were *chota hazree* of tea or coffee and cold toast in the early morning ; a fish breakfast, after my visits, at ten or eleven ; and a hearty English dinner at seven. Most people would require a biscuit and a glass of wine intermediately.

Many years ago my friend, Mr. Charles N. Macnamara, published a short report,¹ which, although many changes have since occurred in the mode of living of European soldiers, contains facts well deserving the attention of every Indian pathologist in the present day. While doing duty for six or eight months with the 1st European Bengal Fusiliers, he made post-mortem examinations in twenty-four cases, and found that there was an abnormal (fatty) condition of the liver and kidneys in all, with the exception of one solitary instance, which was that of a young man who died of an acute disease, after having been only six months in the country. What is even more remarkable, three of these twenty-three cases proved fatal from rupture of large vessels—in one instance of the superior vena cava, in the others of the thoracic aorta—which had become the seats of fatty degeneration. In accounting for this remarkable tendency to fatty degeneration of vital organs among young and vigorous soldiers, Mr. Macnamara showed that, with a European regiment in India, the daily allowance to each man then being a pound of meat, a pound of bread, half a pound of vegetables, and a quarter of a pound of rice, the men added to this most liberal ration one pound of always most inferior meat (often common bazaar pork, afterwards forbidden in General Orders), and one pound of vegetables, and often rice also. So that each man con-

¹ ' Fatty Degeneration of the Liver and other Organs, considered as the chief cause of the High Rate of Mortality among the European Troops of the H.E.I.C. in Bengal,' *I. A.*, October 1855, p. 170.

sumed, on an average, seventy-six ounces of food per diem ; whereas the average consumption by a man of the Royal Navy was then but thirty-five ounces daily, although, in the performance of his duty, he was always compelled to take a large amount of muscular exercise in the open air, whereas the European soldier in India was then in a situation which, unless when on active service, required him to use but little muscular exertion, while he was exposed to the influences of a climate, which, from its very nature, would compel him, if he wished to preserve his health, to live in the most moderate way possible. He then proceeded to show that, while consuming this excessive quantity of hydro-carbonaceous food, the men's systems were, by their manner of living, placed under the most unfavourable circumstances possible for eliminating hydro-carbons (a hot, close atmosphere, want of exercise, and too much sleep), the excess of which must, consequently, be retained in the body. It was said of the European soldiers of that time, by another writer, that, all through the hot days, they did nothing but lie on their beds and curse ! Mr. Macnamara observed, 'If we consider the ordinary routine life of a private in the 1st European Bengal Fusiliers, we shall see how greatly the action of the lungs, as eliminators of carbon, must be impeded. After sleeping through the night in the very close hot air of the barracks, he rises at gun-fire and goes to parade, after which he commonly employs himself in cleaning his accoutrements till breakfast-time (eight o'clock) ; this meal over, he lies down and sleeps till dinner-time ; and after dinner he generally retires to bed again, and sleeps, more or less, till five, the temperature of the barracks being frequently as high as 104° at this period of the day.' He mentioned this particularly, because it is after taking food that the lungs are most active ; if, therefore, heat and sleep diminish their activity, we see at once how small an amount of carbon will be exhaled from them when their functions ought to be least impeded. About five o'clock, the private had to prepare himself for parade ; this over, he sauntered about till half-past nine, and then turned in for the night. Of course there were a few exceptions—some half-dozen to a dozen men taking active exercise every day—and these few were invariably found to be the most healthy men in the regiment. 'The men, then,' Mr. Macnamara added, 'are placed in the same circumstances as animals which are to be fattened ; that is, if we want to fatten an ox, we should keep him in a warm shed, allow him to take little or no exercise, so that no wear of the tissues occurs ; and, owing to the same cause, a minimum of oxygen enters his system, so that the nitrogenised part of the food is stored up as flesh, and the elements of the starch and gum are unburnt, and stored up in the form of fat.' At about that time, Mr. Gant wrote upon the evil results of over-feeding cattle, and there was clearly shown to be a very remarkable pathological parallel between the condition of these

over-fed and under-worked young soldiers, and that of the over-fattened prize-cattle of the London markets. Some years before Mr. Macnamara made the above observations, I wrote :¹—

‘I have long held that several of the forms of visceral disease which destroy most lives among Europeans of all classes in India are—even where nothing amounting to what would generally be regarded as excess, either in eating or drinking, is incurred—mainly dependent upon a system of dietetics which is the very worst and most unphysiological that could possibly be adopted in a tropical climate, that meat, spirits, and beer, which are evidently among the absolute necessities of life among the denizens of a cold climate, where a large supply of highly carbonised material is required by the system, are, although not altogether to be dispensed with, used too largely even by temperate Europeans in India, and that it would be impossible to devise a mode of living more directly calculated to promote that most certain and deadly form of vital decay which determines the formation or increase of fat in the secreting apparatus of the liver and kidneys, and in the muscular tissue of the heart, than that which necessity, rather than choice, imposes on many Europeans during their residence in the East.’

Many and great reforms in the mode of living of our European soldiers in India were inaugurated soon after Mr. Macnamara placed his observation on record. The men were not allowed to make Strasburg geese of themselves, and workshops, schools, reading and coffee rooms, theatres, concerts, lectures, &c., were provided for them, and they were encouraged to spend much of their leisure in cricket, tennis, and other manly exercises. It is still an error in the condition of regimental officers that they have too much leisure in time of peace; still, as a body, they lead active and intellectual lives. To the staff, and to all officers in civil employ, our benevolent Government afford so large a measure of work, under unrelaxing supervision, that it can rarely be said of them that ‘their eyes stand out with fatness, they have more than heart could wish.’

Still, the above observations are full of practical suggestiveness to every medical man now in India, as displaying, in their most aggravated degree, the pathological conditions which a life of excess and indolence would produce in that country, and as throwing great light upon the causation of many otherwise obscure cases of visceral diseases occurring in the present day.

I have never questioned that it would have been better for many of us if we had gone to India vegetarians and teetotalers, and had always adhered to those practices. Still, good food, and beer and wine in great moderation are valid aids to efficient bodily and mental work in

¹ ‘A Practical Treatise on the Management of Diseases of the Heart and of Aortic Aneurism, with especial reference to the Treatment of those Diseases in India.’ (Calcutta, 1851.)

that climate; and I have seen many instances in which injudicious attempts to 'Bant,' and to drop accustomed stimulants suddenly, have led to serious constitutional break-downs. The Government expects us, in giving medical certificates to its officers, to state whether their habits are temperate. In the case of a notoriously free-liver, we best avoid offence by writing 'Habits generous.'¹ A (wine) quart bottle of Bass's pale ale is a fair daily allowance for him who works moderately. He who drinks two bottles is, unless he takes great exercise, as some indigo and tea planters do, a 'generous' liver, who will probably soon fall ill.

Beer, drunk in strict moderation, with abundance of ice, generally agrees well, preventing constipation. A great deal of spoilt bottled ale is always for sale in the bazaars, and is, of course, most pernicious. An officer of Engineers told me that, being on duty with another officer in the bazaar at Cawnpore when the heat was excessive, they asked a petty dealer for two pint bottles of ale. The unhappy man for whom the first bottle was opened drank off its contents at a draught, saying, 'It is vile stuff, but I was so thirsty!' He died of cholera in a few hours. His companion, seeing that the beer was quite muddy and 'gone,' refrained. An old but very healthy lady brought her sick husband by boat to Calcutta for treatment. She was much tried in her attendance upon him. The couple had been in the habit of dividing a wine bottle of beer at dinner. The husband's allowance having been interdicted, she took half the quantity one day, corked the bottle, and drank the remainder on the following day. She was attacked shortly afterwards with fatal cholera. Claret is, doubtless, a wholesome drink in India. It has long been observed that, although acute hepatitis is generally prevalent in the Madras Presidency, the French at Pondicherry, who are, for the most part, moderate claret drinkers, are remarkably free from this malady. But good sound claret is an expensive drink in British India, and a very thirsty man is easily led beyond the bounds of temperance when claret is his beverage.

It is often a matter of serious difficulty to find wholesome *Drinking-Water* in India. Delhi, where the water-supply has always been bad, is notorious for the prevalence of bowel complaints among new arrivals; and Calcutta has the same character. The whole of that vast spread of level country which lies between the Damoodah River, west of Calcutta, and Dacca, on the east, is saline, and none of its water, except rain-water collected in well-puddled tanks, and that taken from its large rivers, above tidal influence, is drinkable. The native patients who crowded my cholera wards in Calcutta were generally strangers from

¹ One of my junior brother officers endeavoured to protect an intemperate patient by writing 'Habits industrious.' The Surgeon-General returned the papers, through me, with one of the sharpest reprimands that it has been my fate to convey.

the country, and I found that most of them had preferred the foul water of the river, wells, or neglected tanks to the excellent water-supply which is now open to all in that city. Very few Europeans are attacked with cholera in Calcutta, with the exception of sailors lately arrived, many of whom, we ascertained, had drunk river-water drawn over the ship's side. Much disease has been found to arise from drinking bad water in hasty marches. It is a general popular belief that paludal fever is often caused by drinking the water of neglected tanks. It is, however, difficult to prove this. I have already spoken of the extreme prevalence of intestinal worms among the native poor in Bengal. Sir Joseph Fayrer's admirable review¹ of modern research into the causes which lead, in tropical climates, to the development of hæmatozoa, is so fully before the profession that it is needless that I should here do more than say that whoever wishes to keep his blood free from *filaria sanguinis* and his integuments and intestines uninvaded by dracunculus and lumbrici, will spare no expense in obtaining his drinking and bathing water from the best sources, and in having them both boiled and carefully filtered before use. For many years, I took these precautions with all our drinking-water (Guinea worm does not prevail in Bengal). The best water procurable was boiled and filtered, and then again boiled, and drunk mostly as weak tea, a bottle of which, slung across the shoulders, affords the best and most refreshing supply on the march.

Native Dietetics.—It cannot be doubted that, in many respects, the diet of rice, wheat, lentils, and oil, used by the majority of the natives of India, is well suited to the climate and to their constitutions. Many Europeans have imitated them in this respect, with advantage, especially during the hottest months. And, most assuredly, the native of India who, upon conversion or change of opinion, begins to live 'generously,' after the manner of some Europeans, is disastrously in error. The great defect in the orthodox Hindu's diet, as well as in the food of the low-castes generally, is its poverty. If it contained rather more animal material, or if the people could afford to allow themselves a larger supply of vegetable oils and good pot-herbs, and plenty of the best fruits of the season, it would be greatly improved; and if they could obtain *ottah* (wheat flour) for one of their two daily meals (which they are always glad to do), the Bengalis would soon rival the up-countrymen in courage and strength. As the potato has impoverished the south of Ireland, so rice maintains perpetual semi-starvation in Bengal. I lost no opportunity of urging the national importance of increasing to the very utmost the cultivation and sale of wheat in Bengal, but it is to be feared that nothing has been effected in this direction, and that our industrious and intelligent brethren, the

¹ 'On the Relation of *Filaria Sanguinis Hominis* to the Endemic Diseases of India,' *M. T.*, February 8, 1879, p. 137, and 'Tropical Diseases,' p. 275.

Caucasian natives of Bengal, will, for many generations to come, go on enduring the miseries of chronic famine upon that very bad and insufficiently nutritious staple of diet, new ¹ *dasee* (common) rice. Physically, the poor rice-feeding Bengali presents a singular contrast to the well-to-do Mohammedan, who eats plenty of flesh-meat and fish. I have before me a Hindu and a Mohammedan skull, very carefully selected in our Calcutta dissecting-room. The difference between these is almost as marked as that between china and crockery-ware, so delicate in structure, so smooth, and almost feminine in character is that of the grain-eater, in comparison with the strong, rough, and coarsely-lined development of that of the flesh-feeder. Here, clearly, the remedy lies with the statesman and the landholder, not with the sanitarian and physician.

Much of the stone in the bladder which prevails so extensively in the Upper Provinces might be prevented if the use of baker's bread (*paon rōti*) could be generally introduced in place of the unleavened chupatties (*hath rōti*) which there form the staple of diet.

As Brahmin cooks are maintained in all our native hospitals, the nourishing food which so large a majority of our patients require, and which we give them freely in accordance with European dietetic rules, is very seldom refused. About once in six months, I found an extremely orthodox Hindu up-countryman obstinately refusing the hospital food. This difficulty was generally met by dieting one of his friends, who cooked for both. Very orthodox Hindus of rank are often very hard to manage, dietetically, in low forms of disease requiring good nourishment. They often refuse milk; and, even if they could be persuaded, in Calcutta, to take the broth of a kid killed by the Brahmins at Kali Ghaut, I have repeatedly seen it produce such unfeigned disgust that I have been compelled to discontinue the attempt. In native hospitals, the full diet is generally a good supply of the best food of the district, including flesh and fish and vegetables, with abundance of milk, and a full allowance of *ghee* (clarified butter) and vegetable oil and spices. Oranges, limes, and other wholesome fruits, are given when needful, and wine and rum are freely employed. In India, the rum must be old; new rum is rendered almost poisonous by the presence of fusel oil. In the low diets, milk, broths, soujee, curds (*dhyye*), and English bread (scarcely to mention sago and arrow-root, in which I have little faith) are most employed.

¹ All rice is unwholesome until after it has been kept for at least a year. The poor cannot afford to store their rice, and so have to eat it soon after it has been cut; the consequence is chronic intestinal irritation.

THE MATERIA MEDICA OF INDIA.

This is a subject upon which the Indian physician ought to acquire considerable practical knowledge ; but it is a matter which lies outside the object of this commentary. Native medical men and patients constantly speak to us of various native remedies with which we ought to be, at least, acquainted, and several indigenous drugs, such as opium, castor oil, ginger, alum, kat-kalija, atees, gurjun balsam, gulancha, ununtamool, chiretta, bael fruit, babul bark, neem leaves for fomentation, mustard oil for liniments, sola for corks, Madras fish oil, telini fly, &c., are frequently used in our own practice. The medical authorities insist upon the free use of indigenous medicines in dispensary practice ; but here it is needful to remark that, if we are not careful to disguise the appearance and flavour of bazaar medicines, our dispensary patients are apt to accuse us of giving them common drugs and of reserving our best medicines for the rich !

It has occurred to most of us, when our supply of European drugs has run short at a remote station, to be compelled to fall back upon bazaar medicines. When I first went to India, we were allowed very little quinine, and had to use bark-powder in our jail and sepoy hospitals. At Chittagong, my supply of bark having run out, and intermittent fever of moderate severity being extremely prevalent, I used the powder of the seeds of kat-kalija, *Cæsalpinia Bonducella* (average dose, twelve to fifteen grains), in cold infusion of chiretta, for several weeks with perfect success. It is related of Dr. William Pitt Muston that, having under his charge 1,500 sick in the force employed against the State of Gopud in 1805, and being without the usual supply of medicine, he relied, 'and with complete success,' on the materia medica of the Gwalior Bazaar.¹

My friend Dr. Waring has brought out three editions of a work which ought to be in the hands of every medical man in India. It is entitled 'Remarks on the Uses of some of the Bazaar Medicines and Common Medical Plants of India,' and is published by Churchill. In this book we are taught, by the highest authority on the materia medica of India, how the drugs commonly procurable in Indian bazaars may, in remote parts of that country, be made sufficient for the treatment of all ordinary diseases if the practitioner be also provided with these *nine* medicines :—Quinine, powdered Ipecacuanha, Calomel, Santonin, Bromide of Potassium, Acetate of Lead, Blistering Fluid, Liquor Ammoniaë, and Carbolic Acid.² The means of studying this very im-

¹ The harm which the absence of mercurials prevented the worthy doctor from doing must not be left out of consideration in estimating the good which the use of bazaar medicines doubtless wrought upon his sick.

² The practitioner ought to make his own blistering fluid from the country

portant subject more deeply are afforded in the Pharmacopœia of India, also prepared by Dr. Waring under the authority of the Secretary of State for India ; in the learned and most carefully prepared work of my friend Odoy Churn Dutt, on the Sanskrit Materia Medica ; and in that great storehouse of Indian pharmaceutical lore, Sir William O'Shaughnessy Brooke's 'Bengal Dispensatory.'

PARASITIC DISEASE.

I have long thought that the parasites—Entozoa, Ectozoa, Entophyta and Epiphyta—which infest the body of man in India afford an ample field for original research ; and, since I left the country, many important additions have been made to our knowledge. The works of the late Dr. Cobbold so fully illustrate all that is at present known with regard to the natural history of those Indian entozoa which produce disease in the human subject, that I shall merely offer a few general notes on the commoner parasites.

Trematoda.

Distoma.—All that is known of the *D. conjunctum*, as found by Dr. McConnell in man in India, and of the *D. crassum* of China, appears in Dr. Cobbold's article Distoma in 'Quain's Dictionary.'

It is mentioned in Gordon's 'China Reports,' p. 206, that *D. Ringeri* was found in the lung tissue of a Portuguese who died of thoracic aneurism.

There is no reason for believing that *Bilharzia hamatobia* has a habitat in India, but it may be expected to occur in men who have served in Egypt and South Africa.

Cestoda.

Bothriocephalus latus, a headless specimen, is preserved in the Calcutta Medical College Museum. In the absence of any history, it must, I think, be considered that the case was imported, probably in a northern sailor, as I know of no reason for believing that cestoid entozoa of this genus are indigenous to India.

Tænia solium and **T. mediocanellata** are only too common in India, and are represented in the Calcutta Medical College Museum. Peet, writing from Bombay experience, says that tape-worm in India is chiefly met with in Europeans, Parsees, Mahomedans, and those Hindoos who make use of animal food. In the Hindoos who abstain from animal food, it is very rare, if it ever occurs. I do not recollect to have seen tape-worm in a Bengal native of any caste, but it is very common amongst the Mussulman population of the Punjab, the lower telini fly, *Mylabris cichorii*; and, if I had my Indian career over again, I would willingly dispense with calomel and acetate of lead.

classes of whom are in the regular habit of eating half-cooked beef—indeed prefer it so.¹ In the four years ending 1883, the returns show only 17, 18, 19 and 24 admissions for tape-worm in our native army; and only 23, 28, 21, and 22 in the jails. I attribute their considerable immunity from this pest to the fact that flesh-eating native soldiers and prisoners take their food well cooked. Now and then, the corner of a soldier's *kiwab* may happen to be swallowed under-done.

Macpherson showed that, in the European army of Bengal, during the four years ending 1853–54, there were, in soldiers, 339 cases or worms, including tape-worm, or $\frac{2}{3}$ per cent. of strength; in officers, 5 cases; in women and children, 10 in each. He adds that many cases in children must have been overlooked. In the four years ending 1883, the admissions were for tape-worm: European women, 5, 7, 6, 15; children, 20, 17, 18, 32; European soldiers, 161, 308, 262, 278; figures which contrast strongly with the tape-worm statistics of native soldiers and prisoners.

Waring met with but one case of tape-worm in Burmah; and an intelligent assistant who had been attached to the local hospitals for nearly fourteen years informed him that he had only once seen a case in Mergui.

Whenever I discovered that a patient had tape-worm, I used to say to him, '*How did you get into the habit of eating raw meat?*' The surprise excited by this question by a stranger was generally great, but the habit was always admitted. I gave full trial to turpentine, kousso, and kameyla. I agree with Dr. Cobbold in giving the preference to oil of male fern, which, of late years, was my only remedy. I found turpentine very efficacious.

Cysticercus.—Some years ago, when travelling, I lodged for some time at one of the largest establishments for 'taking in and doing for' wanderers in the world. A plate of *measly* ham was brought to me. I asked for slices from another joint; these were in the same condition. Having repeated this experiment for a day or two, I advised a professional brother, who sat at the same table, to call for ham from more than one joint and to examine it closely. He at once found that it was diseased meat. I then spoke to my hosts, and, an inquiry being instituted, several uncooked hams were cut, and all were found to be absolutely measly. The oldest servant in the place declared that *he had carved their hams for twenty years, had always found them full of white spots, and had always believed that good hams ought to be thus spotted.* In those twenty years, at least thousands of unfortunate persons, who considered themselves well-to-do, must have battered upon these abominable hams!

Tænia echinococcus.—We have some valuable remarks on the pre-

¹ Oliver—in Aitken.

valence of this parasite in cattle and sheep slaughtered for food in the Punjab, and hydatid cysts and tropical abscess.¹ In the middle of 1869, Dr. J. Fleming called attention to the fact that, from a return by the executive commissariat officer [Meean Meer?], 2,651 cattle were slaughtered in the year 1868 for the use of the troops, and of this number 235 were found cyst-infested, and were condemned, showing a percentage of 8·86. In the years 1866 and 1867, the cattle were shown to have been quite free from cysts. In January and February 1869, 464 cattle were found to be cyst-infested. While Dr. J. Cleghorn was at Mooltan in 1868, several large cysts, taken from the liver of a cow, were brought to him by a medical friend. On examining their contents under the microscope, beautiful specimens of the scolices of *tænia echinococcus* were discovered. He was informed that these cysts were of frequent occurrence in the internal organs, especially the liver and lungs, of the cattle slaughtered in the commissariat yard. He readily obtained permission to inspect the carcasses while they were being cleaned, and, after several months' observation, he found that these cysts were very commonly present. In nine consecutive days, during which 45 cattle were slaughtered, 28 of these were affected with cysts; in one half of these the liver alone was the seat of disease; in 12 both the liver and lungs; in 1 the spleen, and in another the kidney only. Frequent examination detected the presence of scolices in these cysts, and several specimens that were forwarded to Dr. Joseph Ewart, who was then Professor of Pathology in the Calcutta Medical College, were pronounced by him to be hydatids of *tænia echinococcus*. Dr. Cleghorn communicated with commissariat officers in several other stations, and they all agreed in stating that the 'watery bags' were very common in cattle throughout the Punjab; but little attention had been paid to them, as they were considered to be mere 'watery bags' and perfectly harmless. In 899 cattle slaughtered during fifteen months, cysts were found in the liver in 829, and in 726 in the lungs. The great majority of these cases were affected in both the liver and the lungs. In a few the kidneys were affected, and in a less number the spleen.

A most interesting review of this subject has been given by Dr. Aitken,² who found in a sample of beef sent to him from Meean Meer as many as 100 'measle' cysts in the pound of flesh. The medical officer who sent it wrote that he had found as many as 300 in the pound of ration flesh. Dr. Aitken gives an abstract of the report submitted in 1870 by the late Dr. Oliver, R.A., who was specially appointed to inquire into the prevalence of *cysticercus* in the ration beef at Jullundur, which leaves no doubt that both the grazing grounds of the cantonments and the dirty water of a tank there were plentiful sources of tape-worm ova.

¹ *I. M. G.*, vol. iv. p. 114; v. p. 205; vi. p. 41.

² *Op. cit.* vol. i. pp. 205, *et seq.*

Dr. Aitken's observations (at p. 208 *et seq.*) upon the practicability and necessity of stamping out the tape-worm infection which, as we have seen, prevailed at least up to 1883—three years after the publication of the seventh edition of his work—are most just and forcible, as showing the necessity for careful feeding and watering of the cattle, and as throwing strong doubt upon the efficacy of boiling cyst-infested meat; seeing that, although the boiling temperature of 212° Fahr. may be sufficient to destroy the vitality of these cysts, the boiling point is not the temperature at which meat is usually cooked so as to retain the nutritive juices within it; 158° to 170° Fahr. being about the usual range of temperature at which meat is cooked in perfection, so as not to be hard or shrunken.

Echinococcus hominis.—In the chapter on hydatid disease of the liver, I shall give all of importance that I have been able to collect with regard to hydatids as occurring in the human subject in India, and shall discuss the suggestion made by Dr. James Cleghorn 'that a great number of the hepatic abscesses that occur in India may be due to suppurative changes taking place in hydatid cysts previously existing in the liver.' The only case with which I am acquainted in which it appears to be shown that hydatid disease originated in the human subject in India is that of a low-caste woman¹ of Bhopal, in whose spleen hydatids were found. From the fact that this woman was of the sweeper caste, it appeared probable that she contracted the disease from being brought more or less into contact with dogs. I think it not unlikely that this woman may have gone out of India with a European family, as many of her caste do, and so have been infected when on her travels.

If such instances do, in reality, occur, reports of cases of hydatids, in persons who have never been out of India, would be valuable.

Nematoda.

Ascaris lumbricoides.—I have already mentioned that round worms are so common among the poor of Bengal, and especially women, that as a general rule, I made infusion of quassia the vehicle for all fluid medicines in my native female ward. At page 667 of my 'Medical Jurisprudence for India' I have cited the observation of the late Dr. Woodford, whose experience during many years as Police-surgeon in Calcutta was exceedingly large, that 'the pain, low spirits, and weak health produced by the presence of these worms in the intestines, in the rice-eating poor of Bengal, combine as a cause of suicide. Dr. Peet mentioned that these worms are very common among the inhabitants of Bombay.' In the Madras Topographical Reports,² we

¹ I noted this case in the *Indian Medical Gazette*, but have unfortunately mislaid the reference with the reporter's name.

² Centre Division, p. 9.

are told that worms are frequent and remarkably fatal in particular villages in Nellore, being generally believed to be produced by the use of the water of the tanks when nearly dry in the hot season. The disease has prevailed most in the neighbourhood of tanks depending on the rains for their supply. The number of worms found after death is often surprisingly great; persons suffering from them have been known to be seized with the usual symptoms of fever, quickly assuming a typhoid (i.e. typhus-like) character, and terminating fatally in two or three days. In such cases the whole alimentary canal has sometimes been found literally filled with lumbrici.

Old Indian medical writings have frequent references to *worm fever*. I question the existence of such a disease; but it is clear that an ill-fed cachectic wretch whose intestines are full of worms is a bad subject for any severe type of fever. Dr. Neville Jackson published¹ a case which fairly illustrates this point. A man in the bazaar at Balasore had had remittent fever; quinine had been administered with very little effect, the patient still continued to have fever, and was gradually but surely sinking. Latterly, when unable to take nourishment, he complained of a gnawing pain in the epigastrium. There being no apparent organic disease, he was asked whether he had ever passed worms. It was found that he had passed an *ascaris lumbricoïdes* a few days before. It was now suspected that the persistency of the fever and the pain were due to the presence of worms. With this view, four grains of *santonin* were given on alternate evenings. After the first dose, the patient voided one worm; and, after the second, on the fourth day, two more. After this, recovery was rapid, and though he took no more quinine, the fever quitted him.

No more than six cases of *ascarides* appear in the returns of European women for the four years ending 1883. Under the uncertain heading 'vermes,' the admissions of their children were 10, 19, 48 and 23. The admissions of European soldiers were 5, 7, 5, 2. Within my experience, these worms very rarely occur in European men. I never saw or heard of a case in a gentleman. English ladies and children occasionally suffer. Among poor native children, few are exempt. In an old report of the Madras Orphan Asylums,² it is said that these worms were frequent causes of dysentery among the children, who were mostly Indo-Britons. They occurred in vast numbers in several instances; in one the intestines appeared filled with them, and they were found in the stomach and œsophagus. In another case 460 were removed from the body of a small child. When I was in the Cherra Hills, I learnt that these worms are almost certain to appear in anyone who drinks the water of the Cherra Pani—a fine moun-

¹ 'On the Influence of Intestinal Worms in the Causation and Modification of Disease,' *I. M. G.*, vol. i. p. 146.

² 'Medico-Topographical Report,' Madras, p. 63.

tain stream over the gathering-grounds of which the hill-people defæcate.

In some remarks¹ upon the diseases which prevail in the Angami Naga Hills—far northward of the locality above mentioned, Baboo Hemchunder Bhattacharjee notices that round worms are not only frequent in these hills, but also in Assam, where natives and new-comers alike suffer, few being exempt. He adds that the parasite occurs in those who are careful to take water filtered and boiled. I have strong faith in the efficacy of this precaution, but I saw two cases—that of a European lady and her child, in each of whom a large *ascaris lumbricoides* was noticed to be passed once. In that family, it was supposed that none but filtered and boiled water was used; but servants are not always trustworthy; and water containing living germs may sometimes be drunk at friends' houses.

It was observed by the late Dr. A. Simpson, writing from Dacca on stone in the bladder,² that the prevalence of round worms, especially among children suffering from stone, is very great. 'It is,' he adds, 'an exception to meet with one where some of these parasites are not passed before or after the operation.'

In the four years ending 1883, the admissions for round worms in the native army were 50, 47, 43, and 20. Of prisoners 50, 42, 14, and 37 were treated. Doubtless these admissions do not adequately represent the prevalence of round worm in these classes; but they appear to show that natives who are properly fed and lodged and provided with a supply of clean water are not much troubled by these parasites.

In his valuable 'Medical Notes on the Burmese,' my friend Dr. Waring says that intestinal worms (excluding tape-worm), particularly *ascaris lumbricoides*, pervade all ages, grades, and sexes. He found that Europeans and others, after a prolonged residence at Mergui, were no less affected with worms than the native Burmese, although their food was abundant and wholesome, and they had purposely abstained from the use of uncooked vegetables, unripe fruit, and salt fish. It is only these worms which are so common. 'The lumbrici,' he adds, 'are the constant origin of diseases, particularly those of the nervous system.' He has repeatedly seen idiopathic tetanus, epilepsy, chorea, coma, and the most severe spasmodic affections of the bowels caused by these entozoa. After their expulsion the disease has disappeared. Similarly, in the paper cited above, Dr. Neville Jackson narrates interesting cases in which hysterical seizures, passing into epilepsy and dysentery, were benefited when round worms were expelled by the action of *santonin*. In the case of a Brahmin lady, *ætat.* 16, who had been suffering from cholera, but who had recovered from the more urgent symptoms, there was still persistent discharge of *conjee*- (rice-

¹ *I. M. G.*, vol. iv. p. 13.

² *I. A.*, No. 3, p. 64.

water) like fluid, with great prostration. At the same time, she complained of pain in the epigastrium. Santonin, combined with sesqui-carbonate of soda, was given; she passed several round worms, and recovered without another bad symptom. Shortly before Dr. Jackson wrote, there had been an outbreak of epidemic cholera in the Balasore jail. Of eighteen bodies examined, nine contained *ascaris lumbricoides*, and in one of these there were as many as nine worms. Eight of the persons who recovered vomited or voided worms; and, as forty-five persons were attacked, the number of ascertained cases in which worms were noticed was in the ratio of 37·7 per cent.

In the chapter on *Diseases of the Digestive Organs* I shall mention cases in which intestinal difficulty was occasioned by round worms. Two cases of death from lodgment of a worm in the air-passage are given under the heading of *Diseases of the Respiratory System*. Morehead found a lumbricus in a large hepatic abscess occupying almost the entire right lobe of the liver of a Hindoo cart-driver (p. 364). He was disposed to think that hepatitis may be sometimes caused by entozoa. It is undoubtedly *possible* that, in this case, the parasite reached the liver by perforating, and so caused the abscess; but, if this was the case, it appears singular that similar cases have not been frequently observed in India, where these parasites and hepatic abscess are so very common. Morehead found a round worm in the hepatic duct in a case of jaundice complicating remittent fever; he also refers to specimens in the museum at Fort Pitt and at the Jardin des Plantes in which these worms occupy the biliary ducts. To me, it seems probable, considering the great tendency which these parasites have to wander out of the intestinal canal when the sick are dying, that, in the above cases, the parasites had not caused disease in the liver and its ducts.

Practice in India, however, affords numerous striking examples of the *penetrating power of round worms*.¹ Preparation 855 in Ewart's Catalogue shows 'ascarides lumbricoides lodged in the cellular structures between the uterus and the bladder. The fibrous coat of the latter is much hypertrophied and perforated by the worms.' (From a native subject.)

Dr. Waring relates the case of a child, *ætat.* 4, of a Chinaman by a Burmese woman, from the umbilicus of which he saw two round worms expelled. He learnt that the worms had begun to escape in this manner about three days previously, six having already come away. The child was very much emaciated, had been sick for several weeks, and was constantly crying, especially when the worms were expelled. The umbilicus had a red, inflamed, and fistulous appearance; the belly was as hard as a board. Thirteen days subsequently, a considerable

¹ Vide *I. M. G.* for 1871, p. 82, and for 1876, p. 321.

abscess, having appeared about two inches to the right of the umbilicus, was opened and about two ounces of very thick fetid matter escaped. During this interval, the child had continued to pass several worms daily from the umbilicus. Two days afterwards, two large worms passed by the incision. Up to this date, twenty-four worms had escaped through the abdominal parietes. The child was very much emaciated. Under the use of sulphate of iron, five or six worms were passed per anum. Dr. Waring did not see the child after this; but was informed that, under the use of theet-sai (a native vermifuge, an exudation of the melanorrhœa usitatissima, used for lacquering boxes, images, &c.), a large number of worms were voided naturally, and that the child perfectly recovered.

Ascaris mystax.—I do not believe that this parasite is indigenous to Lower Bengal. Shortly before Dr. Cobbold described it in 1868, Dr. S. C. Townsend and native doctor Meer Enayet Hossein sent from Nagpoor to our museum specimens which were probably of this kind.¹ A Mussulman child, 1 year and 9 months old, had been passing worms for some time before he came under treatment, but was otherwise in good health. Three stools were produced by a vermifuge dose. The mother said that she counted upwards of 3,000 living worms, which she burnt, thinking that this measure would kill those that remained. On the third day, a dose produced four stools. With the first was ejected a mass of 775 worms, which Doctor Townsend counted; with the second 100, and the third 440. On the following day, the child suffering considerable pain in the bowels, another dose brought away 250 and 100 worms. 'The next morning 440 worms were passed in one stool.' The mass was brought to Dr. Townsend and counted before him. In the course of that day 800 more were passed. The next day nearly 200; and, on the next, 60. After this, a few were passed daily for two or three days, and then the child became free from them. The mass containing 775 worms and weighing nearly four ounces, was sent to the college museum. The reporter described them as being 'like thread-worms, pointed at each extremity, and quite white, but they measured from two to seven inches in length, and weighed from one to seven grains.' The officiating curator considered that they were 'probably a small variety of the *ascaris lumbricoides*, as they resemble the usual form of that worm in all respects, save that they are only a third of the normal size.' The people of Nagpoor, Dr. Townsend adds, for the most part draw their drinking-water from wells, which are overshadowed by large trees, and which contain much organic matter. *Ascaris lumbricoides* is extremely common among all classes; but, as far as he could ascertain, the kind above described was not known.

When we say, 'drink none but the very best water afforded by the

¹ Case of worms—species unknown—treated in the city of Nagpoor. *I. M. G.* for July 1, 1867, p. 181.

locality, let that be filtered and boiled, and again let it be drunk only as weak tea or soup,' we recommend the whole prophylaxis of round worms. The words 'santonin, followed by a course of quinine, quassia, and iron,' sufficiently represent our therapeutic measures. Dr. Waring says: 'In one or two cases of severe abdominal spasm, I have deemed it advisable, as a temporary measure, to administer a full dose of the tincture of opium, and subsequently a cathartic; and, in every instance in which this formed the treatment, the worms were expelled lifeless, as if poisoned by the large dose of the opiate.'

I do not find that *Trichocephalus dispar*, as occurring in man, is represented in our college catalogue. I only recollect to have met with this species once in India (it was perhaps imported), although I had seen cases in London.

Prep. 540 in the college museum catalogue is the liver of a common [ship?] rat with a cyst in its centre to which a trichocephalus dispar is attached. There are two other cysts containing remnants of similar dead worms.

Trichina spiralis.—I was present at the post-mortem examination in which, as Dr. Cobbold mentions, Mr. Hilton first recognised 'the calcified and lemon-shaped capsules ("gritty particles").' The only other case of trichinosis which occurred within my observation was detected in Calcutta by Sir Joseph Fayrer in a stout and singularly florid European in the prime of life, the subject of very extensive malignant disease of the shoulder. The returns show that, in 1881, two European soldiers were admitted in the Bengal Presidency for trichinosis, one of whom died; and in 1882 a criminal in the Punjaub suffered from the same disease. There was also one case in the jails in 1881.

Filaria Medinensis.—In his valuable chapter on 'Dracunculus' Morehead gave a table showing that in the eight years ending 1834, there were 390 admissions in the 4th Light Dragoons between 600 and 700 strong, at Kirkee. He also showed that, in 1832 and 1833, the European soldiers of the Bombay Presidency (who in the former year were 5,184 strong) had 262 and 60 admissions for dracunculus. In the former year at Kirkee only there were 206 admissions. In the following year there were only 24 admissions at that station. Dr. Joseph Ewart has shown¹ that Matoongha, in the Bombay Presidency, was 'abandoned many years ago by the head-quarters of the artillery on account of the prevalence of Guinea-worm in both officers and men;' and that, in the eleven years ending 1839, a total of 55 admissions for Guinea-worm took place in an aggregate strength of 103,431 European soldiers in the *Madras* Presidency.

A great triumph—shall we say of sanitation or of awakened common

¹ Vital Statistics of the Meywar Bheel Corps, *J. A.*, No. 12, p. 452.

sense?—appears in the Sanitary Commissioner's Returns for the four years ending 1883, in which it is shown that the totals of cases of Guinea-worm in the whole of the *European Army* were in Bengal 1, Madras 10, and Bombay 14.

In the four years ending 1883, the admissions of *Sepoys*, in all India, were 762, with one death; 1,247, with five deaths; 396 and 432. Among *Prisoners* the yearly admissions were 765, 512, 401, and 469. One year's (1882) admissions of native soldiers will fairly illustrate the distribution, *as regards Locality*, of this pest. There occurred in the *Bengal Presidency* 40 cases, in the *Punjab Frontier Force* 22, in the *Central India Force* 165, in the *Hyderabad Contingent* 56, in the *Bombay Army* 142, and in the *Army of Madras* 47. A careful analysis of the above data would, I am confident, show that the sepoys of the Bengal and Madras armies who suffered were stationed *westward*, thus (1) the majority of the *Bengal* native soldiers were probably men of the Meywar Bheel Corps. Thus Dr. Irvine wrote in his 'Topography of Ajmeer,' p. 118: 'Cases of Guinea-worm are extensively scattered over the west of India, but in no place is it more frequent than in Marwar.' In the report cited above, J. Ewart has shown that at Kherwarrah and Kotrah (the worm is rare at Kotrah), between 1841 and 1858, an aggregate of 2,682 cases of Guinea-worm occurred in an aggregate of 161,831 native soldiers, or about 1 in 6. An aggregate of 170 Europeans had not a single case. (2) A very large proportion of the *Madras* Sepoys were probably attacked either in the vicinity of the city of Madras or at Secundrabad.—*Vide* Dr. Van Someren's 'Summary of Cases of Dracunculus,' treated in the hospital of the 15th Native Infantry at the latter station in 1852—'Proceedings of the Hyderabad Medical Society.' Dr. Waring cites¹ the 'Madras Topographical Reports'² to the effect that, previous to the year 1834, the town of Madras appears to have been singularly exempt from Guinea-worm. It is shown above that the greatest prevalence of Guinea-worm at Kirkee in the Bombay Presidency, when every third European in the 4th Light Dragoons was attacked, occurred two years previously, in 1832. It is said 'Guinea-worm has been observed to occur annually for the last six years' (previous to 1842) 'in the villages of Vepery, Chintadrepettah, and Perambore (part of the township of Madras). It was not seen in any of these places prior to that period.' Dr. Waring gives a table showing the annual number of cases treated at the dispensary of the second of the above-named 'villages' in each of the fifteen years ending 1848. They were 5, 8, 23, 119, 67, 133, 88, 86, 24, 56, 80, 273, 63, 223, 281—a total of 920 treated in the fifteen years. In the report of the Mysore division, p. 120, it is said: 'Dracunculus is very uncommon in this country, and, of the 170 cases,

¹ 'Notes on Some of the Diseases of India,' *I. A.*, No. 6, p. 500.

² Madras, p. 17.

with one death, seen in the table, no fewer than 72 occurred in the 18th Regiment Native Infantry in the first half-year of 1838.' This corps had been stationed at Madras during the early part of the previous year, in a locality where the disease is known to exist; and, as the time the filaria takes for its development in the human system is supposed to be twelve months, it must have entered the bodies of these men whilst stationed at Madras in the months of February, March, and April, 1837. I never saw, or, as I believe, heard, of a case of dracunculus originating in Lower Bengal or in any part of Eastern India.¹ With regard to the North-West Provinces, Dr. John Jackson, now of Brighton, remarked,² writing at Ghazeepore, that all the cases of dracunculus that he had seen in that part had been previously living in the south, especially on the Bombay side. He was not aware of the occurrence of any cases between that station and Calcutta.

Mr. F. H. Brett³ wrote upon this condition as it prevailed in the valley of the Dhoon, among the cultivators of the district and the grass-cutters and *sacess* (grooms) of the Body Guard.

Ewart observes that the prevalence of Guinea-worm has been associated with the existence of trap rocks. This fact was first pointed out by Dr. Chisholm in Grenada, where the wells were sunk in trap rock. Its extreme rarity in localities where laterite abounds has been noticed by Morehead and Waring. On the west coast of Africa, Dr. Horton found the parasite in the greatest abundance in places where trap rocks of volcanic origin were undergoing decomposition.

In commenting upon the sudden outbreak of the parasite in the suburbs of Madras in 1839, Dr. Waring states his opinion that the disease 'may be transplanted from place to place, and that localities previously exempt from the disease may become the seat of this affection.' Dr. W. J. Moore observes⁴ that, in Marwar, cattle, camels, and dogs are occasionally found afflicted with a parasite which appears precisely similar to the common dracunculus.

In his article on Guinea-worm in 'Quain's Dictionary,' Dr. Cobbold held that the notion that Guinea-worm finds ingress to the human body through the ducts of the skin—as where negroes and sepoy go about bare-footed in swampy places, and school-boys bathe in a pond swarming with microscopic tank-worms (Carter) must now be abandoned. He cites Fedschenko to the effect that the escaped embryos of the worm perforate the skin of minute crustaceans (cyclops); here, after a period of only twelve hours, the embryos undergo a fresh change

¹ Cases appeared in the Calcutta General Hospital in Twining's time, but the subjects were foreigners, Arab merchants.

² *Cal. Med. and Phys. Trans.*, vol. viii. Part 2, Appendix, p. 314.

³ *I. M. J.*, vol. v., N. S., p. 491.

⁴ 'Marwar, the Land of Death,' *I. A.*, No. 20, p. 525.

of skin. At the expiration of one month and six days, they acquire their highest larval stage of growth within the cyclops, and then, along with the intermediate hosts, as young males and females, they are transferred to the human stomach, where, or in the intestine, they copulate. The males then perish and the females migrate through the tissues to the surface of the body. The above opinion that the parasite gains admission to the body of man by means of the drinking-water is a very old one. Dr. Kennedy stated that he had learnt from the natives that their belief, through all ages of which they have record, has been that 'the disease is occasioned by the generation under the skin of a living animal whose larvæ are deposited there externally, or drunk with water.'

Mr. Brett, writing in 1840, mentioned that the natives of Deyrah attribute this condition 'to the worm contained in the water which is drunk from the various streams permeating the valley.'

In his account of Marwar, Dr. Moore says that 'the people are strongly convinced that the ova of this pest are introduced into the system with the drinking-water. Dr. Smyttan, of the Bombay Establishment, wrote: 'It has been satisfactorily proved that these animals find their way into the body only through the stomach, the ova being contained in the water drunk.' Dr. Chisholm admitted that, while the worm may sometimes enter the body through the skin, it may be received into the human stomach, in its embryo state, by drinking the water of certain wells.

A very careful, practical sifting of the questions, Does Guinea-worm originate from the tank-worm? and Does it enter the system by the skin? was made by Mr. Henry Martineau Greenhow while in medical charge of the Mairwara local battalion. The result of his inquiry convinced him that both questions must be answered in the negative. Although the developed parasites usually occur in the lower limbs, Dr. Smyttan believed that 'they had been found in all other parts of the body, as far as the cellular membrane extends.' In the body of a gunner, he found a full-grown Guinea-worm attached to the peritoneal covering of the liver, and another, which was alive and writhing, attached to the peritoneum over the left kidney—otherwise they floated loose among the viscera. Dr. Bird, as cited by Waring, mentions the case of a man 'who died from some other disease' in whom a Guinea-worm was found on the surface of the liver. Dr. Scott, also cited by the above authority, mentions having known the worm residing in the eye-socket, in the mouth, in the cheeks and below the tongue. Dr. Ewart mentions that, during seventeen years' residence at Kherwarrah, his native doctor had only seen one worm issue from the orbital cavity, and two worms, in separate individuals, made their

¹ On *Dracunculus* in Mairwara, *J. A.*, No. 6, p. 556.

exit from under the tongue. He adds that such cases are extremely rare. Dr. Horton describes it as occurring rarely, in African negroes, in the breast and testicle.

In a Mussulman drummer, who was syphilitic and died of Bright's disease (Morehead, p. 479), a Guinea-worm which had undergone cretaceous change, was found between the inner surface of the middle lobe of the right lung and the pericardium and connected to both surfaces by adhesions. Waring found that, in 132 cases, 125, or about 94 per cent., were situated in the lower limbs.

Although generally indisposed to move after it has attained full growth and has taken up a position beneath the skin, the worm has, in rare cases, shown considerable power of migrating from one part of the body to another. Dr. Smyttan observed this in the cases of two officers. In Lieutenant F. the worm could be distinctly traced under the skin at the top of the left shoulder; by-and-by it found its way to the elbow, where it was as distinct; and, in the course of a few weeks, it made its way, by gradual progress, to the wrist, where it was extracted. In the case of Lieutenant S., the worm was first observed under the skin on the inside of the biceps. It found its way to the elbow joint, and by-and-by was very distinct half-way down the fore-arm. Here it remained stationary for some time, and at length retraced its course to the inner condyle of the humerus, where it was extracted in an extremely lively state. Dr. L. W. Stewart, of Madras,¹ gives the case of an officer who had extracted a worm fifteen inches long from his scrotum. 'Ten days afterwards, he experienced an unpleasant sensation in the posterior aspect of the left thigh. Day by day the sensation shifted lower down till it reached the popliteal space. A few days later it was experienced in the calf. Hitherto nothing was visible, but, at the end of sixteen days from the first sensation in the thigh, the convolutions of a Guinea-worm could be distinctly traced at the outer side of the ankle-joint. Dr. Stewart now wished to extract it, but the evening was too dark, and he delayed until the following morning. Then, however, the parasite had entirely disappeared, having, it was considered, taken up a position among the deep muscles of the foot. Many abscesses formed, with severe inflammation of the foot, which confined the patient for three months before he was free from this wandering parasite.'

A crowd of observers attest the fact that the development of the worm in the human body occupies a twelvemonth. I do not find any Indian authority for Dr. Tilbury Fox's statement that the latency of dracunculus is about six or seven months.²

Ewart has shown, on ample statistical data, that the half of the year comprising the hot and rainy seasons is the period when dracun-

¹ Cited by Dr. Horton.

² *Lancet*, March 8, 1879.

culus abounds, abruptly beginning at the commencement of the hot season, and terminating, more abruptly still, with the exhaustion of the monsoon in September. In the seventeen half-years ending August, there were 2,909 admissions; in the seventeen half-years ending February, there were only 373; the proportion being more than 6 to 1. He adds that these data correspond in the main with the records of the Bombay Army given by Morehead. In like manner Dr. Moore observes¹ that, in Rajpootana, there is 'a seasonal maximum of the disease, occurring in the months of June, July, and August.' Waring found that, 'at Madras, dracunculus annually appears with great prevalence during the hot season; at Bombay and Matoongha, during the monsoon or rainy months; at Dharwar, during the latter portion of the hot season and the commencement of the rains; whilst at Sattara, it is observed chiefly during the rains and towards their termination.' He adds that, 'it is a remarkable fact that it invariably manifests itself as an epidemic at the same period of the year, in all the above-named localities. Isolated cases may, however, occur at any season or period of the year.' The Abbé Dubois stated that, in the central parts of the Madras territory, it prevails in December, January, and February.

With regard to the *causes of death* in the few fatal cases of which full reports have been given, Dr. Ewart says that, of the two deaths which had occurred among the men of the Bheel Corps, Dr. Bowhill stated that 'one died from exhaustion produced by the copious discharge, the other from the abscess, occasioned by the presence of the worm, having burst into the abdominal cavity.' These were the only fatal cases recorded out of an aggregate strength of 16,831, extending over a period of upwards of seventeen years. Dr. Bowhill stated that 'a camel-man died in consequence of the extreme sloughing.'

Mr. Minas relates² the case of a native, *ætat.* 30, a farmer. 'The whole body and the skin was a network of Guinea-worm; a portion of the entozoon used to come out, break, and cause considerable local suffering. Abscesses, sinuses, and afterwards sloughing ulcers formed, with high irritative fever. The repetition of these so exhausted the strength of the patient, that he died on the eighty-fourth day after admission.'

At Ajmere, Meer Ushruf Alee saw only one fatal case in upwards of 200 treated. The patient died from exhaustion produced by the profuse discharge from an abscess in the thigh.

Dr. Ewart cites Dr. Leith's 'Deaths in Bombay,' where it is shown that 133 deaths from dracunculus and its consequent suppurative inflammation occurred in that city during eight years. In the chapter

¹ *I. M. G.*, October 1874.

² 'Notes on Dracunculus, with the Plan of Treatment adopted by the Natives.' *I. A.*, No. 6, p. 568.

on tetanus, I have cited many cases in which that disease was caused by the irritation of a Guinea-worm.

Treatment.—It is clear that boiling the water used for drinking and bathing and also filtering the drinking-water is a needful precaution wherever this parasite abounds. As Dr. James Johnson long ago observed: ‘Mercury, carried to the length of impregnation of the system, has been considered by some as a specific, and so has assa-fœtida; but the local means are those most to be depended on. *Sublata causa*, &c.’ I should not have made the above citation, seeing that, as I believe, all Indian surgeons practise extraction, but for the fact that Dr. J. Horton¹ recommends assa-fœtida as a radical cure for Guinea-worm; saying that this drug appears to have a direct influence on the worm by at once destroying it, dispelling the pain and enabling the individual to walk about with ease and freedom. My late excellent friend Meer Ushruf Alee, who had large experience of dracunculus in Ajmeer,² wrote: ‘Assa-fœtida has been much esteemed by natives as a prophylactic. In my opinion, this medicine and pure water for drinking are the best prophylactics for this disease.’

Although an English surgeon, who happened to be afflicted with Guinea-worm, would probably far rather be treated by a brother officer than by a native, however famous for these cures, some of the worm-extractors seem to possess considerable skill. A gentleman, writing to the ‘I. M. G.’³ from Poonah, said that, when a Guinea-worm first protruded its head from his leg, the end was tied to a piece of lint, and the worm reeled off, in the orthodox manner, an inch or two morning and evening. After about ten inches had been thus extracted, the worm got broken, and the end was lost. The limb swelled and became very much inflamed. Fomentations were continued for ten days without any benefit. Then a dirty half-naked man was sent to him from the Gunesh Khind Bazaar. He had a lancet and a copper tube, about five inches long, shaped like the frustum of a cone. He enlarged the wound a little, and then, having applied the large end of the tube over it, commenced sucking violently at the other end. After keeping up a vacuum for about five minutes, he let in the air, withdrew the tube, and there were twenty inches of the worm, looking like so much boiled vermicelli, in a mass of coagulated blood. The reporter adds: ‘My leg has been getting better ever since.’ This was very probably a conjuring trick, the worm remaining untouched.

An old officer told Dr. Kennedy that, when on a march during the first campaign against Seringapatam, a Seedy (African) stepped out of the ranks and, pointing to a rapid stream of water, requested permission to go thither in order to relieve himself of a worm in his ankle,

¹ *The Diseases of Tropical Climates*, &c., p. 639.

² *I. M. G.*, vol. iii. p. 6. ‘A Few Practical Remarks on the Treatment of Guinea-worm.’

³ Vol. v. p. 171.

after a manner that he had seen practised in his native country. He loosened the worm, of which part had been extracted, from the cloth round which it was secured, and plunged his naked foot into the current. The force of the running water was sufficient to release the worm almost immediately. Upon this case, Dr. Kennedy founded a mode of treatment; and, up to the present time, a steady stream of water is employed as an aid to moderate sustained traction.

Filaria oculi.—See Diseases of the Eye.

Filaria sanguinis hominis.—See Elephantiasis, and Chyluria. I believe that the typical strongyles have not been observed in India.

Oxyuris.—Oxyurides occur in India much as they do in England.

In his paper on the Diseases of Burmah, Dr. Waring says that, at Mergui, 'these worms are comparatively rare. Where you would meet with one case of thread-worm, you would meet with a hundred of *A. lumbricoides*.

Assistant-Surgeon Bhowani Das considers that oxyuris may be a cause of fistula in ano.

Insecta Parasitica.

For cases of idiopathic ozæna, of syphilitic disease of the nose and of otorrhœa, in which maggots of the *musca carnaria* infested the passages, see the chapters on Diseases of the Nose and Ear. Dr. J. Butler Hamilton¹ admitted an artilleryman with severe pain and inflammation of the foot. There was a very small orifice close to the nail of the fourth toe at its outer side. Movement being detected in a black speck just visible in the opening, a lively maggot, about half an inch long, was removed by forceps, and then two others. The hole was then filled with carbolic oil and cotton. That evening, five more of the same length came away. Cotton dipped in pure carbolic acid was introduced, and next morning two came away. Under the same treatment three maggots, each fully an inch long, came away. Recovery was speedy.

Dr. A. Garden has observed² that it has happened to himself twice, once in case of amputation and once after lithotomy, to find the wounds filled with maggots in the intervals between two morning visits; of course, through the carelessness of the attendants. He adds: 'Maggots in the nose, in wounds, in the human frame, are simply the result of carelessness and want of cleanliness. They are matters quite accidental and secondary to the original disease. Keep out the flies and the part cannot become fly-blown.'

¹ *I. M. G.*, vol. vi, p. 36.

² Remarks on Ozæna, *I. M. G.*, vol. v. p. 107.

Ectozoa and Arachnida Parasitica.

Pediculi, *Sarcoptes scabiei*, and *Demodex folliculorum* occur in India nearly as they do in Europe; but I do not know that their structure has been examined specially. Has Malabar Itch an acarus?

Suctoria Parasitica.

I have given elsewhere cases in which *leeches* were found lodged in the nose, larynx, fauces, and urethra. I know, by personal experience, at Chittagong and far northwards of that place, in passing through the *terai* at the foot of the Kasia Hills, that *wood leeches* and *ticks* are sadly annoying to travellers in jungly tracts. The leeches look like little withered sprigs standing out stiffly from the bushes along the way. By a powerful muscular effort, they throw themselves backwards upon the hands of the European and the bare legs of the native passers-by. I did not notice the ticks until I found little red blebs on the backs of my hands—these are the bodies of the wretches distended with the blood which they have sucked.

Dr. W. Palmer notices that Europeans with delicate skins, who walk on Calcutta grass in the rains or autumnal months, are liable to experience a severe itching between the ankles and knees, where a number of raised, flat-topped bright red papules are seen thickly studded round the part immediately above the top of the boot. These, in a small proportion of cases, run into pustules and ulcers which heal with difficulty, leaving pigmented scars. These are caused by *sand flies* (*pulex penetrans*). The bites of these insects or of fleas or mosquitos probably cause those little irritable ulcers about the ankles which constitute *Bursati*. I generally found that these got well under rest and nitric acid wash.

The 'I. M. G.' for January 1885 has an account of the *Peepsa* fly of the Hills of Assam, by Drs. Brauer and Beecher. In his report of the Aka Expeditionary Force, 1883-84,¹ Surgeon-Major E. R. Johnson states that, among the troops, the admissions on account of the bites of these insects were only exceeded in number by those due to ague and dysentery. The flies, he says, attack the hands and feet; they are noiseless, operate very quickly, and cause no pain at the time. The puncture soon causes a conical elevation of the skin, capped by a red blister, the size of a pin's head. This blister soon becomes of a deep purple black, and the place itches for many days. As the insect does not attack parts covered with clothing, the hands and feet suffer most; and, when the bites are numerous, these become puffy and œdematous, blisters and bullæ sometimes form, and small scratches are converted into unhealthy sores. These have no specific character, and, under

¹ S. C. R. for 1883, p. 169, Appendix A.

appropriate treatment, rest and protection from further attacks, soon heal. It is added that, when once a man's feet were swollen, it was found better to send him back at once to the field hospital where the flies were not found. Mr. Ordham considers that this insect is identical with the *Poto* of the N.W. Frontier.

Mosquitos.—Apart from the grave suspicion which attaches to them of causing filarious disease, these insects are generally very troublesome to new arrivals, especially to those who are full-blooded and of fair complexion. The recruits, of whom I had medical charge in my first voyage to India, were inspected naked by a medical board soon after landing. One big fair man (who was afterwards hanged for murder) had his entire surface so completely set with large papules, each caused by a mosquito, that the end of a pen-holder could not have found space between any two. When one has been long in India the spots are generally petechial, rarely papular. The lividity of these and of flea and bug bites is often, as in England, an evidence of impaired health. I repeatedly noticed that, while the mosquitos in my own house spared me, probably having experience of the fact that I did not afford wholesome nourishment, I generally suffered miserably from these creatures when I had to sit up at night in strange houses. The natives are subject to mosquito bites. In winter, this leads them to smoke the pests out of their houses with such determination that one is almost suffocated in driving through the streets at night. Coming up hastily to the bedside of a very dark native in my ward, I saw his forearms so closely beset with papular mosquito bites that, at the first glance, I thought of small-pox.

Persons bathing on the sea-coasts of India are liable to suffer great skin irritation, which is attributed to contact with sea-nettles, &c. Dr. John Meredith relates¹ the case of a gentleman, who, being heated, rode his horse through a tank in the Chupra district, which, as he afterwards learned, is avoided by the natives on account of its water not being good to bathe in. Immediately after landing, he felt painful itching all over his body. The horse was in a like state, immediately beginning to rub, roll, and bite itself. This went on for several days with but little rest, until a great part of the hair was rubbed off its body, and its skin was sore in many places. About a fortnight afterwards, the greater part of the man's body was seen covered with nail-marks, as in cases of prurigo. He had noticed minute vesicles emitting a small quantity of glairy fluid, very irritable but not sore. In Assam, Dr. Meredith noticed cases of irritation and itching of the body attributed to the character of the water used in bathing. I think it very probable that the water was pure enough, and that the eruption was due to going in while heated and then drying in intense sun-glare.—*Vide* 'Sun-Burn,' under the head of Cutaneous Diseases.

¹ *I. M. G.*, vol. vi. p. 130, Caution to Bathers.

Caterpillars frequently run over the skins of children, leaving their hairs set in the skin. The native servants remove the hairs by a little apparatus made by tying a thread tightly round an end of the child's hair, which is then cut off about an eighth of an inch above the thread. Thus a little stiff flat-ended brush is formed—this being used tweezer-like, the hairs are easily drawn out.

At pp. 386–87 of my 'Medical Jurisprudence for India,' I have given some information with regard to the effects of the *stings of bees, wasps, hornets, and scorpions* in India. I published some additional facts on scorpion stings in the 'I. M. G.' for April 1874.

Mr. Alexander Grant has mentioned that wasp and bee stings may often be extracted by pressing the mouth of the barrel of a key over the puncture.

Scorpion sting is occasionally fatal in children. I have a note (unfortunately without the reference) of a reported case of death in 12 hours from scorpion sting. The sufferer was a female grass-cutter aged 14 years, but immature. Apothecary Goggan reported two cases.¹ The first was that of a Hindoo cart-driver, *æt.* 40, whose death followed the accident in three hours and a half. The second case appears questionable. An apparently strong Hindoo, *æt.* 55, died with hemiplegia three days after the accident. The reporter understands that the rock or ground scorpion, supposed to be small and of a reddish colour, is liable to cause death.

My friend Surgeon-General Hugh Macpherson has lately confirmed the observation that the application of chloroform speedily relieves the pain of scorpion sting. I have a note, by Dr. Shortt, of Madras, to the effect that scorpion sting does not harm the large house lizard (the ghecko) which feeds on scorpions. He adds that it is said that the best remedy for scorpion sting is to slit up a lizard and apply it to the injured part. (I never saw but one ghecko in Bengal.) He tried it once in a young lady with perfect success. My friend Dr. Francis Day tells me that it will always be noted that the skin immediately surrounding the part stung by a scorpion perspires.

In 1872, the 'Delhi Gazette' reported that Mr. Strachan, the Superintendent of Cotton Experiments in Scinde, had found carbolic acid to be a good remedy for scorpion sting. 'He had treated seven cases with it. Four were workmen, and taken at once' (I conclude this means whenever the application was made immediately) 'they did not stop work for an hour.' In August 1879, carbolic acid was also recommended in the 'British Medical Journal' (p. 267) as an antidote for bee stings. An English lady, on waking in the morning, felt great smarting at the back of the neck, upon which were some red irregular streaks such as might be caused by strokes with a large

¹ I. M. G. for March 1884.

camel's-hair brush moist with an irritant fluid. It is said that such painful marks are made when a small house-lizard, common in Bengal, which is alleged to be poisonous, runs over the bare skin. The tarantula and the centipede are the only other house-pests likely to cause such lesion.

Entophyta and Epiphyta.

Sarcina Ventriculi.—See 'Diseases of the Digestive System.'

Chionyphe Carteri, Mycetoma.—On this subject I have nothing to add to the well-known writings of Vandyke Carter and Lewis and Cunningham.¹ I saw the case reported by Sir J. Fayrer at p. 637 of his 'Clinical and Pathological Observations.' Its subject was a Moham-medan agricultural labourer of the Burdwan District, about eighty miles north-west of Calcutta. It is very remarkable that, as far as I am aware, no other case of this disease (which, if it occurred, could hardly be overlooked) has been reported as observed in Lower Bengal. Western Burdwan is out of the Gangetic Delta, and its soil is not alluvial. As far as we know at present, this locality may be taken as the western boundary of the habitat of mycetoma. A valuable summary of the early history of observations on this very singular disease has been given by Surgeon-General Moore.²

Mycetoma is not named in the returns for 1880 and 1881. In 1882 one sepoy was admitted; in that year a total of three, and in 1883, two prisoners were treated.

Animal Poisons.

Sir Joseph Fayrer's great work, together with the researches of Dr. Joseph Ewart, Dr. Vincent Richards, and Dr. Wall, completely occupy the subject of *Snake Poison* in India.

Poisons Employed in India.

In my 'Medical Jurisprudence in India' I gave full notices of those poisons which are generally employed by Indian criminals. I regret that I have not been able to embody, in a new edition, numerous important facts which I have since accumulated.

VARIOLA, VACCINIA, VARICELLA.

The learned research of Dr. T. A. Wise¹ has shown that the Hindus and also the Chinese were familiar with small-pox many centuries before it was described by the Arabian physicians. It, however,

¹ Article 'Fungus Disease of India' in Quain, which contains at p. 521 a full reference to Dr. Carter's numerous published observations on this malady.

² *I. M. G.*, vol. ii. p. 269.

³ 'Commentary on the Hindu System of Medicine.'

is not probable that Hindustan was its original source—unless its cradle was among the aboriginal hill tribes—as it appeared suddenly in the plains of India, and committed such ravages that it was named after the goddess Shitalá, at whose shrine those attacked vow to make offerings, and whose ministers were the earliest inoculators. Dr. Wise holds that the pest was probably carried westward by the Persian conquerors of Hindustan; and that, as navigation extended, ships from India conveyed it to the Arabian ports of the Red Sea, where it seems first to have appeared A.D. 900.

From time immemorial, variolous inoculation has been employed by the natives of India. This practice, as carried on by the *teecadars*, is considered to be agreeable to Shitalá, the goddess of small-pox, to whom vaccination is held to be offensive. Recklessly and unscientifically as it is generally carried on, often surreptitiously in evasion of the law, and directly operative as it is in the maintenance and diffusion of small-pox, inoculation is still, in India, as it was in England previous to the use of vaccination, a very important protective among those who obstinately reject vaccination. Thus, in 1854, my friend the late Dr. Bedford showed, from data collected by himself, Dr. Wise, and Dr. Wilson, that, among 33,811 inhabitants of three large towns in Bengal Proper, 82·12 per cent. bore the cicatrix of successful inoculation on their forearms.² Considering all that has been done with a view to extend vaccination in Bengal during the three decades which have followed Bedford's inquiry, it appears almost impossible to resist the belief that, at least in the most carefully vaccinated districts, the above high percentage must have now become sensibly diminished; nevertheless, the Surgeon-General's reports show that, in the gaols of the Burdwan Division, in 1872-73, the percentage of inoculated in 1,406 individuals was 85·21 per cent. The difficulties which attend the advance of vaccination, especially eastward in Bengal, and still more signally in Orissa and in the greater part of the Province of Assam, have led some to question whether Government ought not to abandon vaccina-

¹ The native inoculators of India can hardly be more ignorant and reckless in practice than were some of those who inoculated the English poor in last century. I copy the following extraordinary notice from the *Oxford Journal* of February 4, 1758:—'I George Ridler near Stroud in the county of Gloster Broadweaver at the desier of peeple hereabout do give nautis that I have inokilated these too seasons past between 2 and 300 for the small pox and but two or three of them died. A mainy peeple be a feard of the thing but evaith it is no more than scrattin a bit of a haul in their yarm a pushin in a peece of skraped rag dipt in sum of the pocky matter of a child under the distemper. That every body in the nasion may be sarved I will God willin undertake to innokillat them with the pervizer they will take too purges before hand and loose a little blud away, for half a crown a head; and I will be bould enough to say noo body goes beyond me. N.B.—Poor volk at a shillin a head but all must pay for the purgin.'

² *I. A.*, No. 1, p. 183.

tion and to carry on inoculation systematically, as they formerly did in their European regiments. The arguments in favour of systematic inoculation in India are fully stated at page 124 of the admirable work of my late friend Dr. Kenneth Mackinnon, 'A Treatise on the Public Health, Climate, Hygiene, and Prevailing Diseases of Bengal and the North-West Provinces,' which, although published in 1848, is still invaluable to the Indian practitioner. While fully admitting the self-evident objections to this measure, I have always doubted whether its adoption would not have effected more good than that of vaccination has done.

Let us add to the above facts the following suggestive data:—In the examinations described by Dr. Bedford and in the Surgeon-General's reports, as cited above, it was found that the proportions of those who had undergone vaccination, in 1854, were 5·6 per cent., and in 1872–73, none. It must be admitted that the practical validity of these last figures is diminished by the facts that the first set of observations was made thirty-two years ago, and that the subjects of the second were of the ignorant criminal class, among whom the benefits of vaccination are least likely to be recognised. It was, however, shown in the Surgeon-General's reports for Bengal Proper, that, although the annual proportion of persons requiring protection was 2,049,000, the annual number of vaccinations was only 365,622 in 1871–72. and 687,406 (500,094 fully verified) in 1872–73. The same authority has shown, that, at least up to 1869, the amount of protection conferred throughout the whole of the Bengal Presidency was altogether insufficient, as vaccination was received by only—

22·35 per cent. of the children born in Bengal.				
27·14	“	“	“	N.-W. Provinces.
36·47	“	“	“	Punjaub.
38·46	“	“	“	Central Provinces.
3·63	“	“	“	Oudh.

My friend Dr. Kenneth McLeod added that, taking the whole of these provinces, affording, at 3 per cent., 3,490,146 births, only 878,654 were successfully vaccinated, or 25·17 per cent. In 1884 the operations in Bengal had increased to 1,488,292—more by 123,624 than in 1883–4 ('I. M. G.'). In the Madras Presidency, vaccination goes on with activity, but is still susceptible of great extension. The report of sanitary measures in India shows that, in the year 1876–77, the number of vaccinations in that Presidency was 483,192, an increase of 101,780 on the results of the previous year. Dr. Cornish rated the success at 94·33 per cent. of those vaccinated. In 1883, there were 749,146 operations. In his inaugural address¹ in the Epidemiological Society, Surgeon-General John Murray showed that, in the three Presidencies, the number of vaccinations in 1875 and 1876 was only 3,729,275, of which

¹ *M. T.*, November 28, 1878, p. 606.

3,390,007 were successful. The statement of moral and material progress of India for 1881-82 shows that the total number of vaccinations in all India, in that twelvemonth, was 4,414,342, with a percentage of success to primary operation of from 80 to 98·23 per cent., an increasing result which, highly creditable as it is to the vaccinators, falls very short of affording due protection to a population of nearly ninety-five millions, among whom the annual birth-rate must range between 3 and 4 per cent. Inoculation is still carried on to a great extent, especially in the Lower Provinces. It has never been very prevalent in Upper India. Throughout the Province of Assam, unsanctioned inoculation is practised as a rule. At Gowalparah, Assam, in 1871, the priests fined and put out of caste individuals who had their children vaccinated. The people of the Province of Orissa appear to entertain an unconquerable aversion to vaccination. To extend the law prohibiting inoculation to any district is self-evidently a measure which requires many considerations, and the existence of a strong probability that the inhabitants may be brought to accept vaccination, and to discard inoculation. Thus, in Assam, vaccination was, a few years since, distinctly practised to a very small extent, but appears to be gaining ground. About eight years ago Dr. De Renzy¹ reported that variola was endemic in Assam, the indigenous population suffering very severely; but the coolie population of the tea-gardens were entirely exempt, as vaccination is compulsory on them. In 1883-4 there were 88,693 operations against 45,246 in 1881-82 and 24,179 in 1880. In Orissa, vaccination has yet to be accepted. In his returns for the four years ending 1883, the Sanitary Commissioner only mentions this province twice (in 1880 and 1881), alluding to 'increase' and 'considerable decrease.' Sir J. C. Brown reported that the districts of Bengal, where inoculation is not practised, or on the decline, are—Calcutta and Midnapore, very little inoculation; Darjeeling Circle, not so universal; Arrah, diminished; Monghyr, Bhaugulpore, and Sonthal Pergunnahs, much diminished; Chota Nagpore Division, seldom practised. In 1866, Dr. Aitchison induced the Hakeems of Umritsur to practise vaccination.

It has been deemed advisable of late to employ the services of the native inoculators in vaccination. My friend Dr. Chandra, when Civil Surgeon of the Sonthal Pergunnahs, in 1869, reported: 'The inoculators like the work, because it is less troublesome and less risky than inoculation, and the people themselves appeared to be satisfied and happy.' Subsequently, in 1873, Sir J. C. Brown stated that much progress had been made in the instruction of inoculators, although it had been practised only in the districts of Bancoorah, Midnapore, Kamroop (in Assam), Patna, Arrah, Chumparun, Monghyr,

Bhaugulpore, Sonthal Pergunnahs, Singbhoom, Maunbhoom. He suggested that, to facilitate and perfect the system, a subsistence allowance ought to be given to the men while under instruction; moderate rewards should be assigned to those who do really well; and, above all, systematic inspection should be provided. In 1880, these men appear to have performed 803,545 operations.

In 1869, the Surgeon-General reported that, both in Calcutta and Bombay, the reproduction of strong virus through the calf is needful, owing to the degeneration of the lymph by vaccination from arm to arm during the hot and rainy seasons. Not so, according to Dr. Watson, in the North-West Provinces, where abundance of good virus can be supplied from the children of the Sub-Himalaya Mountains. Later, in 1873, this authority held that there appeared to be no reason why the Darjeeling Hills should not be used as nurseries for the lymph required to set vaccination a-going in the plains; as the hills of Kumaon and Gurhwal, the Tebree and Murree Hills, are successfully employed for the purpose in the North-West Provinces and Punjaub. In 1873 my friend, Dr. Charles, Superintendent-General of Vaccination, Calcutta, reported that the stock of virus originally taken from the calf in Calcutta, which had never previously been through the human system, had, on March 31, 1873, reached its 142nd transmission through man. It had retained its efficacy unimpaired, but was in no way superior to the old stock of lymph which was still in use in other parts of the town.¹ The Calcutta vaccinating season may be said to begin from the month of October. It is recommended to practise the operations as little as possible during the hot months in the plains. In the hills it succeeds better. Not only is vaccination likely to fail if attempted during the wrong season, but, as was shown by my friend Dr. T. W. Wilson,² vaccination among natives of Bengal, towards the end of the rainy season, often produced irregular appearances, being followed by papular and vesicular eruptions. Indeed, Dr. Wilson, a very cautious and accurate observer, held that—‘By continuing to use the same stock of lymph a disease identical with small-pox is the result, having all the worst features of that malady, being infectious and often fatal, and these secondary eruptions are not of rare occurrence.’ On the occurrence of such accidents it used to be popularly rumoured that our vaccinators spiced their vaccine lymph with variolous matter.³ I recollect a very painful and in some respects

¹ It is reported that, in 1876, small-pox was very prevalent in Bombay, and vaccination became popular. ‘Morning and evening a vaccinator with a heifer was sent round the streets, so that vaccination was brought to the very doors of the people.’ In Bengal Proper, there were 32,273 direct and indirect bovine vaccinations in 1883.

² ‘On Varioloid Eruptions consequent to Vaccination in Bengal,’ *Indian Register of Medical Science*, 1848, p. 541.

³ In his annual report upon vaccination at Bassein in 1866–67 (*I. M. G.* for

inexplicable case in which a young native gentleman of some importance died of small-pox after having been vaccinated by one of my native doctors. The patient had come in to the civil station, in some alarm, from a village where small-pox existed. If, however, he caught the disease there, the period of incubation was excessively prolonged. I nevertheless had not the slightest reason to doubt the perfect good faith of my native doctor, to whom the mishap was a sad blow.

The question of the liability of the vaccine disease to degenerate in India is discussed fully, and, considering that the author was an advocate for inoculation in preference to vaccination, very fairly, by Dr. Mackinnon;¹ but as, under an improved system, the reports of the existence of an ineffective virus, except when the operation is attempted in the hot and rainy seasons, have become rare of late, I shall not say more upon this question, in which it must always be impossible to found accurate conclusions upon other people's cases.

As in Europe, it is a matter of general observation that, both in the North-Western Provinces and in Bengal Proper, small-pox may be expected to appear late in the cold season and to prevail most from January until May, greatly declining or entirely disappearing in the rains. My friend, Dr. John Macpherson, has shown² that, during a term of twenty-nine years, the deaths from small-pox in Calcutta occurred as follows:—In January, 1,425 deaths; February, 2,845; March, 4,934; April, 4,249; May, 2,261; June, 1,054; July, 555; August, 223; September, 188; October, 147; November, 132; December, 576. It appears clearly in Dr. A. H. Leith's 'Death Statistics of Bombay,' that the same law is operative in Western India. In the five years 1852–56 the death-rate from variola generally began to rise in December. It was high and increasing in January and February. It was very high in March, and reached its maximum in April. In May it fell considerably, but was still very high. Thenceforward there was a progressive decrease; but, in June and July, the deaths were numerous. In the four succeeding months, the death-rate was generally low. In the September and October of 1854 and 1855 there were not any deaths from this cause.

In the absence of perfectly reliable census returns, our measure—August 1, 1867), Mr. A. Callaway Nisbet writes:—'Many have confessed to me that after applying for lymph—as a blind, it would appear, to escape some fancied punishment for practising inoculation—and after having had the arms of their children duly punctured in the prescribed fashion—nay, after the operation had been duly performed by the unsuspecting Government vaccinators—small-pox virus had been subsequently introduced into the punctures, and, as I observed myself in many cases, inoculation in the Burmese fashion had been employed likewise at one and the same time.'

¹ 'On the Epidemics of the Bengal and North-West Presidencies,' *I. A.*, No. 3, 1854, p. 147.

² *Cholera in its Home*, p. 4.

ment of the prevalence of small-pox among the natives is not very exact. Bedford's statistics, already noticed, show that, among 33,811 persons, 389 had suffered from natural small-pox. Out of 1,406 prisoners in the jails of the Burdwan division, 105 had also suffered from the disease. Of course these statistics only represent the survivals. Nearly 3,000 deaths from small-pox occurred in Calcutta in February and March, 1865. In 1872-73 there were only nineteen deaths from small-pox in that city. In the five years 1852-56 the numbers of registered deaths from small-pox in Bombay were 1,007, 649, 313, 1,014, and 199, in a population which was estimated at 566,120 in 1849.

Dr. John Murray showed from the returns, in 1875 and 1876, that the mortality from small-pox in India is reckoned at nearly 200,000, while, in the two preceding years, it was nearly half a million. In 1883, there were 232,363 deaths, against 85,131 in 1882.

Inquiries made by the Medical Board, and cited by Dr. Kenneth Mackinnon, showed that, amongst the entire Madras Native Army, for ten years ending 1888, in an aggregate strength of 568,403, the cases of small-pox recorded numbered only 415, and the deaths 42.

According to Dr. Kenneth Mackinnon, the strength of the European troops in Bengal, during the six years from 1846-47 to 1851-52, averaged 18,795. In that period there were 229 cases of small-pox, of which 65 were fatal. Recent reports, as cited by Dr. Murray, give a much more favourable result. During 1875 and 1876 only two deaths resulted from this disease among 120,000 European troops systematically vaccinated. In 1883, there were 105 European soldiers treated, with 9 deaths.

The profession in India have always been remarkable for their promptness in adopting the newest scientific discoveries. The Royal Jennerian Society was established in London in 1803; and, in the following year, Dr. Shoolbred reported 1,426 successful vaccinations in Calcutta. The first superintendent-general of vaccination in Bengal was appointed in 1816. In 1873 the staff at that centre was composed of a superintendent-general, 3 superintendents, 11 deputy and native superintendents, 404 vaccinators, 24 apprentices, and 272 ex-inoculators who practised vaccination on their own account. In 1883, there were 1,131 vaccinators at work in Lower Bengal.

As we have shown above, small-pox generally appears in Calcutta and Bombay towards the end of each cold season. Its rise in those cities appears to be principally attributable to the activity of the inoculators and to the movement down country of itinerant traders from the north. At intervals of from four to six years, it rages in the native city as a pestilence. At these times, when the contagion is at its fiercest height, it, in many cases, defies all protection. I have seen cases in persons deeply scarred by a previous attack. It was probably

upon the occasion of such a visitation that Dr. Macpherson¹ found that, out of thirty cases of confluent small-pox treated by him in one year in the Calcutta General Hospital, twenty-five had been vaccinated, and twenty died.

Poor food, the influence of paludal poison, evil drainage, and imperfect ventilation in crowded city dwellings, often render the pest cruelly destructive, especially in the young, in whom the corneæ and joints frequently suffer. I have before me the photograph of a Calcutta beggar, the foundation of whose entire face is one cicatrix, destitute of features, save that the mouth is a lipless chasm, and that the eye-balls roll under the new integument which has formed completely over them. Quinine would probably have prevented this destruction. We can notice very little essential difference between the small-pox of India and that of Europe.

Dr. Duncan Stewart, as cited by Dr. Mackinnon, stated, as the result of his great experience in Calcutta—first, that, in seasons of epidemic small-pox, *Varicella* is not seen; second, that, on the other hand, in a season when chicken-pox shows itself, we need not fear an outbreak of small-pox during that season. He says: ‘When a well-marked case occurs in a school early in the season, I have no fear of small-pox appearing also that year.’ Elsewhere, however, the two diseases (if they be wholly distinct maladies) have been noticed to prevail synchronously.

Varicella is not very prevalent in India. Mackinnon found that, in the three years 1849–52, among the children of the European soldiers in Bengal, averaging 2,291, there were only twenty-four cases of chicken-pox during the whole period, and no deaths. In the four years ending 1883, the admissions were 58, 39, 18, and 18.

MORBILLI.

Measles runs nearly the same course in India that it does in England, save that, among the children of well-to-do Europeans, it is possibly less dangerous there than it is at home.² Still, this disease is very common and very destructive among the children of the native poor in cities. Morehead proved that ‘when we compare the history of measles in India with that of the disease in colder climates, we find the mortality to be much higher in the former. The rates of mortality stand thus: European countries, 3· per cent.; Byculla Schools, Bombay, first epidemic, 5·; second, 3·4; third, 5·4; fourth, 8·5; Moreshwar Junardun’s cases, 18·; Infant Schools, Poona, 22·5;

¹ Cited by Dr. Mackinnon.

² Dr. Mackinnon observed that ‘It is a very principal difference in the diseases of Indian children how little mortality they suffer from the exanthemata, small-pox excepted, and even this has reference to native children.’

1st Battalion Artillery, Nuggur, 28; Bengal and N. W. Provinces, 8. In 1868 it was reported by Dr. Charles, Superintendent of Vaccination, that Calcutta had been visited by an epidemic of measles, which was widely spread throughout the city, and in many places seems to have affected almost every house. Dr. Leith's statistics show that, in the five years 1852-56, the deaths from measles in Bombay (the population of which was calculated in 1849 at 566,120) were 106, 131, 130, none, and 164. Highly valuable data with regard to the history of measles in Bombay and Western India have been given by Drs. Morehead¹ and J. H. Carter.² The returns for the four years ending 1883 show that, during that term, 37 *European women* were attacked with measles; of these one died. 123 *European soldiers* were admitted, and there were 3 deaths. The admissions and deaths of *Soldiers' Children* were 335, 11 died; 441, 32 died; 231, 1 died; and 84, 2 died. *Native troops*, 563 admissions, 13 deaths. *Prisoners*, 361 admissions and 8 deaths. Many years ago, when the women and children of European regiments were too often lodged in hot, ill-ventilated, overerowed barracks, and badly clothed and fed, measles, when it appeared among them, which was not very frequently, did great mischief. Dr. Murphy³ reported such an outbreak among the children of H.M.'s 80th at Dinapore, who appear to have been placed under very unfavourable hygienic circumstances. There were no less than 120 cases and 6 deaths out of a strength of 165.

Dr. Duncan Stewart and Dr. John Macpherson have shown that measles is usually epidemic in Calcutta in January, February, and March. In the Upper Provinces, the researches of Mackinnon show that it generally occurs later, at the hottest season. In this respect, it follows the course of cholera, appearing in the North-West after it has almost ceased in Bengal. In the report cited above, Dr. Carter says that the disease attacked 107 children in a school averaging about 330 inmates. Six cases proved fatal by dysentery, diarrhoea, pneumonia, marasmus and fever, while three other casualties from dysentery (2) and marasmus and fever (1) occurred under circumstances which suggested the idea that the infection of measles might have had something to do with their fatal termination. In reviewing this report⁴ at the time I remarked: 'Our own Indian experience leads us to regard measles, especially when occurring in the middle of the cold season, with much more serious apprehension than is entertained by the majority of authorities in this country. We have learned to view every case of dysentery, diarrhoea, and thoracic affection, occurring during the prevalence of measles, with great suspicion. Nothing can

¹ *Clinical Researches on Disease in India.*

² 'Medical History of the Central Schools of Bombay,' *Trans. Med. and Phys. Soc. of Bombay*, No. 1, N. S., 1853.

³ Cited by Mackinnon.

⁴ *I. A.*, No. 1, 1854.

be more certain than the fact that one or other of these affections may constitute, from first to last, the only marked feature in an attack of measles, coryza being altogether absent, and the skin remaining smooth and white, until excited by a warm bath or by mustard applications, when little more than an outline of the, not retrocedent, but undeveloped eruption presents itself.' My own sad experience has taught me that, in this condition, especially in anæmiated children, severe renal, laryngeal, and endocardial, as well as pulmonary and intestinal complications may occur. At pp. 198-99 of his great work, Dr. Morehead said that, in an epidemic of measles which attacked the Byculla Schools in 1838-39, he examined the five fatal cases. In all there was pneumonia, which, in four, had passed on to hepatisation, with, in one, gangrenous excavations. In all, there had been muco-enteritis, which in two had led to granular exudation on the mucous lining of the lower part of the ileum and of the colon; in one to turgescence and ulceration of Peyer's glands; in one to redness of the mucous membrane of the lower part of the ileum, and turgescence of Peyer's glands; and in one merely to vascularity of the end of the ileum. He considered that there is probably greater danger in measles from gastro-intestinal inflammation in India than in the same disease in more temperate climates. And he held that, as an additional cause of high mortality, the greater obscurity of pneumonia in asthenic states and the less control over its course are deserving of notice. In the hot season, brain mischief is always threatened. In the epidemic in the 80th Regiment, alluded to above, the heat being very great, head complications, often attended by convulsions, were prevalent, the six deaths were so caused. There appear to have been no cases of serious chest complication.¹

Dr. Macgregor,² the field of whose experience was principally the North-Western Provinces, dwelt upon the fact that the sudden transitions of temperature from heat to cold, so frequent in all parts of India, should be carefully guarded against in measles. He argues with justice that, when measles is prevalent in the cold months, the infection should be avoided as much as possible, since the cold will tend to aggravate the disorder, and to cause dangerous chest (and bowel) complications. On the other hand he urges that, when measles prevails in February (when the weather is warm), there is little or no use in avoiding it, for the disease is one which people must undergo once in their lives,

¹ Dr. F. P. Staples published in the *I. M. G.*, February 1, 1872, p. 30, what appears to have been a typical example of the Morbillose Blut Dissolution described by Dr. Franz Mayer. It occurred in a European soldier, and was characterised by symptoms of extreme poisoning of the nervous system, with a bluish colour and early fading of the eruption, and an enormous deposit of albumen in the urine.

² *Practical Observations on the Principal Diseases affecting the Health of the European and Native Soldiers in the North-Western Provinces of India*, 1843.

and it is better that the trial should be got over when young. He especially insists that children should not be removed to the hills in the state of debility which follows measles, at least until a considerable interval has elapsed, and until the hot weather has set in at the hill-stations. Measles has not unfrequently broken out among the children in troop and passenger ships, on the voyage round the Cape, so long after leaving land as somewhat to countenance Dr. Salisbury's view that the fungi of wheat-straw are the source of measles.

EPIDEMIC ROSE RASH.

At a meeting of the Epidemiological Society held in February 1885, Dr. Kenneth McLeod read a paper on the occurrence of Epidemic Roseola in Calcutta.¹ In the year 1880 and the early part of 1881, an epidemic of measles visited that city. This was succeeded by an outbreak of cases of an eruptive fever, occurring sporadically throughout the town, among all classes, but principally children and young adults. It lasted throughout the hot weather and rains of 1881, and cases continued to occur during the cold weather of 1881-2. The symptoms and course of these cases differed in many respects from those of the epidemic of measles. This was the opinion of the medical men from whom Dr. McLeod, as health officer, sought information, as well as of the natives, who called the disease *minnina*, the term *hām* being usually applied to measles. There was always a distinct pre-eruptive stage, generally lasting for from twenty-four to thirty-six hours, ushered in by chilliness, malaise, anorexia, &c., and characterised by the sudden onset of smart fever. Muscular pains were sometimes present, vomiting was rare, and watery eyes, running nose, and sneezing not common or severe. The eruption was papular in spots and patches of circular (not crescentic) shape, sometimes becoming diffuse or confluent, bright red, rosy or crimson, appearing generally, but not always, successively on face, body, and extremities, lasting for two or three days, and not succeeded by desquamation. The fever was in most cases severe, reaching from 100° to 105°; its onset was sudden and its decline gradual, as the eruption faded. The total duration of the illness was about a week. The most commonly observed complications were bronchial catarrh and tonsillitis. The fauces and palate were always congested, and the tonsils sometimes much swollen, especially towards the close of the outbreak. They were sometimes seen to be covered with an aphthous pellicle, and slight ulceration was also observed. The lymphatic glands of the neck were enlarged in many cases; diarrhœa was rare. The urine was not albuminous, and no signs of dropsy were seen. Sequelæ were absent, and the mortality was trifling.

¹ Vide *Trans.* of the Soc., N. S. vol. iv. p. 52.

Fatal cases were described, but it was doubtful whether the deaths were from this malady. The disease was not very infectious, but cases occurred in succession in the same family, and instances of communication by personal intercourse were related. It was observed that a previous attack of measles was not protective, and a case was recorded in which an attack of measles occurred shortly after recovery from this disease. Dr. McLeod considered that its phenomena and course resembled those of rubeola and r  theln more closely than those of any other exanthem. It was held that there could be no question of the identity of this malady with scarlatina, because, in addition to well-marked clinical differences, it is a well-established fact that scarlatina does not occur in India as an epidemic. Isolated cases resembling scarlatina have been observed, and the disease has been repeatedly imported by troop-ships, but it has always died out. The author showed, by a careful analysis, that the evidence pointed strongly to the conclusion that this fever was no other than r  theln. Mr. E. F. Drake-Brockman reported what he considered to be an outbreak of r  theln in the town of Madras in 1876. It is noteworthy that, as Dr. McLeod was reading this paper, in the second week of February 1885, cases of epidemic rose rash were occurring in Bayswater.

FEBRIS RUBRA.

I never saw any form of scarlatina in Lower Bengal, or any disease which could be fairly mistaken for it; neither has any medical man with whom I have discussed the subject met with a genuine and unmistakable case in that great province.¹ Writing in 1864,² Dr. Peet, who had large experience in the Bombay Presidency, said: 'Scarlet fever is altogether unknown in Western India.' According to Dr. William Anderson, of Yedo, Japan is entirely free from this disease.³ Dr. R. G. Daunt⁴ states that scarlatina was first recognised in the interior of Brazil in 1881. It having been reported that a case of scarlatina had appeared at Simla in May 1870, the editors of the 'I. M. G.,' with a view to place the question of the occurrence of true scarlet fever in Calcutta in a definite position, addressed a circular to all medical men practising there, calling for an expression of their opinions and statement of their experience on the subject. A summary of the replies was published in October 1871. There was in these a remarkable unanimity on the negative side. Not one of our

¹ The opinion of my friend, the late Dr. Chuckerbutty, that the so-called Dengue which prevailed in Calcutta in 1871-72 was scarlatina, will be considered under the head of Denguis.

² *Principles and Practice of Medicine*, p. 539.

³ Dr. Dobell's *Reports on Diseases of the Chest*, vol. iii. p. 14.

⁴ *M. T.*, January 27, 1883.

body had seen a typical case of scarlatina in India. There were perhaps some isolated cases which appeared to have been imported from Europe. Dr. F. N. Macnamara had seen four by no means indisputable cases in an Armenian family. Most of us had considerable personal experience of Dengue, a disease perfectly distinct from pure scarlet fever.

Still there cannot be a doubt that the celerity with which passengers and letters¹ are now conveyed to and from Europe and India has become a means by which an interchange of dire maladies—foremost among which is scarlatina, formerly kept apart rather by time and distance than by any essential difference in climate—is becoming gradually but certainly effected.

That scarlatina is, from time to time, conveyed from Europe to India, especially in troop-ships, is now an established fact.

In 1864, Dr. Peet mentioned that scarlatina had occurred in some of the military stations of the Madras Presidency, soon after the arrival of ships with the families of soldiers, the disease having prevailed on board. But he was not aware that the disease had spread to the native population, or had continued to prevail amongst those by whom it was imported.

The next appearance of scarlatina in India was announced to the profession at home in the 'Lancet,' February 11, 1871, and also by Dr. Louis Thomas, who stated² that it has been proved that scarlatina first appeared in Iceland in 1827, and in Greenland in 1847 and 1848, and gradually extended over large tracts of land in Asia, and lastly, 'according to Maunsell and Cunningham, also attacked India, which had enjoyed immunity from it for a long time.'

The occurrence here alluded to was reported by Assistant-Surgeon Maunsell, of the R. H. Artillery, in the 'I. M. G.' for December 1870. In the previous May, this officer encountered at Simla what he was fully satisfied was a case of scarlatina. For my own part, I am not at all persuaded by Mr. Maunsell's brief narrative that the case of Captain E. was one of scarlatina. It is mentioned that the urine was not examined for albumen.

Two months subsequent to this occurrence, Dr. A. Garden had, in August 1869, at Saharunpore, a case of what he termed 'Erythema Scarlatiniforme.'³

Dr. Garden was judiciously cautious in accepting this case as one of scarlatina. He asked: 'In an epidemic of scarlatina, would not this

¹ Dr. Cornelius Fox stated in the *Sanitary Record* that there can be no question that the poison of scarlet fever is disseminated by letters, for proofs of this accident have been repeatedly afforded. The outbreak of this disease in village post-offices had presented itself to his notice on two occasions, and in both cases the disease spread.

² Ziemssen's *Cyclopædia*, art. 'Scarlatina,' vol. ii. p. 160.

³ *I. M. G.* for July 1870.

case be accepted as a fairly typical case of the mild form ?' But he added that it wanted one of the chief factors of that disease—viz. contagiousness. Though it occurred in a house occupied by two large families of children, no others were affected. Although considerable pressure of argument was laid upon him, Dr. Garden steadily declined to admit, in face of the fact that he had never seen nor heard of an undoubted case in India, that this isolated case (unattended as it was with the slightest evidence of a tendency to spread in a family of eighteen or nineteen persons, most of whom were children) was an unquestionable instance of pure scarlatina.¹

Definite information regarding the introduction of true scarlatina into India was to follow.

In August 1871, Dr. G. J. Gibson, of the 1-2 Queen's, reported in the 'I. M. G.' that in Poonah, during the months of May, June, and July 1870, thirteen cases of genuine scarlet fever occurred among the children of his regiment. Dr. Gibson gives selections from his case-book which strongly support his conclusion that all the symptoms and sequelæ of scarlatina were present. The rash always existed, appearing on the second day. The tongue, after a few days, presented the strawberry condition. There was the inflammation of the tonsils, pharynx, parotid and submaxillary glands. There were discharges from the nose and ears, and one child recovered perfectly deaf. Desquamation occurred in all the cases (in two the nails on the hands and feet were renewed). During this period, dropsy and albuminuria appeared in six cases, the outbreak having occurred during the monsoon (stormy) season, and it being difficult to keep the children in bed : Dr. Gibson several times found those recovering standing in the verandah with nothing on excepting a small nightdress.

Where the first child caught the disease was unknown, but it was reported that there were two cases of scarlatina in the Ordnance Lines. It is to be regretted that we have no note of the date on which the last draft arrived at Poonah. This point is, however, of minor importance, seeing how very short and easy the transit from Bombay to Poonah is.

In the 'I. M. G.' of September 1871, the following very important observation was made by Dr. Lundy, of H.M. 58th Regiment :—On February 1 of that year, a draft, 122 strong, arrived at Bombay from England. It was afterwards discovered that the disease had appeared on the voyage out, and that two men and four children of the draft were left behind at Aden, as they were too unwell to proceed, and might besides propagate the disease on board. On March 3 the draft arrived at Sealkote. With them were three married families, the children of which mixed freely with the others. On March 30 three children were admitted, and it very soon became apparent that they

¹ I. M. G., October 2, 1871.

were suffering from scarlatina. Eleven cases were admitted. The first four were of a mild type; the remainder being more or less severe. Two cases were fatal; and, from the symptoms, Dr. Lundy considered them to be scarlatina maligna. The remaining cases had the characters of scarlatina anginosa, the febrile symptoms being attended with pain; enlargement and suppuration of the submaxillary glands; bright redness of uvula and tonsils; back of throat coated with thick mucus; great pain and difficulty in swallowing; tongue swollen and red, papillæ raised; inflammation of the mucous membrane of the nose, and the usual eruption. In the nine cases that recovered, there was desquamation of the cuticle, unaccompanied, except in two, by any severe irritation. Œdema of the extremities formed a prominent symptom, as did also albuminous urine. The pulse ranged from 103 to 127; the heat of skin from 102° to 109°.

Soon after the draft arrived, two or three men of it were admitted with what was then put down as a feverish attack. Subsequently, Dr. Lundy was inclined to think that they, in reality, suffered from that very mild form of scarlatina which often escapes detection. Indeed, one of the men was, shortly after discharge, re-admitted suffering from anasarca.

Another draft of men who went out to India in the same infected troop-ship, proceeded in March to join the 92nd Regiment at Jullundur. About a month afterwards scarlatina broke out among the children, and some of them died.¹ Towards the end of December 1871, it was reported that the 'Jumna' had arrived at Bombay, and had been placed in temporary quarantine in consequence of the prevalence of scarlatina and measles on board.

The facts and arguments adduced by Staff-Surgeon Bradshaw and others, fail to convince me that scarlatina has ever been known to originate *de novo* in India. But since he, Dr. Garden,² and Dr. R. D. Murray³ consider that they have seen it so originate there, all practitioners have doubtless remained on the watch for its appearance. Its tendency to spread and to establish itself in that country does not appear to be by any means strong. It is self-evidently most to be feared where it occurs in the cold and rainy seasons and at the hill stations. To prevent its admission to the country, and to stamp it out at any station where it may present itself, ought never to be a matter of difficulty wherever vigilance and care are observed.

I know of no evidence of its existence among the civil native population of India.

The report of the sanitary commissioner with the Government of India for 1871 gives 110 admissions and 17 deaths from scarlet fever among the children of the European army in India :—Dinapore, 1

¹ Staff-Surgeon A. F. Bradshaw, *I. M. G.*, August 1, 1871.

² *Ibid.*, May 1, 1872.

³ *Ibid.*, May 1, 1876.

admission; Meean Meer, 37 admissions, and 6 deaths; Sealkote, 12 admissions, 1 death; Jullundur, 19 admissions, and 3 deaths; Umballa, 1 admission, and 1 death; Deolalee (Bombay troops), 1 admission, and 1 death; Aden, 17 admissions, and no death.

In the four years, 1880-83, there occurred, throughout India, the following admissions and deaths, under the heading 'Scarlet Fever': *European soldiers*, 8, 8, 19, 2 admissions, no death. *European women*, 2, 0, 1, 0 admissions, no death. *Soldiers' children*, 11 (3 deaths), 16 (2 deaths), 9, 10. *Native soldiers*, 1, 1, 0, 2. No case occurred in jail during the four years.

BERIBERI FEVER.

I am led by a careful inquiry into the ætiology of Beriberi, and especially by a review of the history of the epidemic which visited Bengal and Mauritius in 1877 and 1878, to regard this malady as a specific fever, *sui generis*; and, consequently, to be of opinion that physicians will remove it from its present anomalous position in our nomenclature of disease, as the last and least defined in the roll of general diseases, and place it next to scarlatina, with which fever it has certain marked features in common. Since this remark first appeared, the College of Physicians have placed 'Beriberia' next to 'Febris Malarialis.'

We are told¹ that the word Beriberi is derived from the Hindustani name of a sheep (*b'here*), from a fanciful notion that persons affected with the disease walk like a peculiar species of that animal, *i.e.* with a kind of jerking motion; but the name by which it is known to the Gentoos of the coast, *Ooboo-waioo*, simply means rheumatism combined with dropsical swelling.² The writer adds, 'This disease, though endemic in many situations, frequently also appears as an epidemic after the setting in of the rains, or from July to the close of the year, when fevers, dysenteric complaints, and cholera also break out, to a greater or less extent annually.'

Morehead justly objected to the unscientific vernacular designation of this malady; but the name beriberi has been so long accepted that it would now be useless to attempt to abandon it entirely, although our present knowledge of the ætiology of the disease renders it needful that we should modify it—as by calling it 'beriberi fever.'

The form of acute general anarsarca, generally recognised under the name of beriberi, occurs as an endemic and as an epidemic. Its principal endemic habitats are Ceylon and portions of the Madras

¹ *Madras Topographical Reports*, Russel Condah, p. 89.

² Those who may be desirous to trace out fully the etymology of beriberi will find ample data in Fayer's article, Quain's Dict. p. 104, and in vol. ii. O. S. of the *I. M. J.*, p. 343.

Presidency. According to Hamilton, the limits of the Indian *endemic* were the Malabar coast and that tract of country which extends from Madras to Ganjam, being confined to these parts, and extending no farther inland than forty miles. Later observation has considerably extended this endemic area, the limits of which it may have transgressed in recent times. Its ascertained *epidemic* range is exceedingly wide; its field occupying a very large expanse of the globe's surface—the South Pacific Ocean and its islands; the southern part of Australia and Mauritius in the southern hemisphere, and, in the northern hemisphere, as high as the Kasia hills, above the parallel of 25°. If we are to regard the epidemic which invaded Lower Bengal, in 1877, as true beriberi, as I think we must, the influence of this malady, when it becomes epidemic, may be expected to extend, in a plain country, as far as the sea breeze does, *i.e.* about 250 miles inland—Calcutta standing 110 miles, and Shillong some 140 miles from the sea. Both places suffered in the recent epidemic. Dr. Feris shows that, invading Brazil at Bahia in 1864, beriberi ‘soon spread to provinces remote from the sea-board.’

In his report on the kakké—the beriberi of Japan—Dr. Duane B. Simmons gives a map from which, considering the fact that this disease occurred in H.M.S. ‘Juno,’ at Sydney, after a lengthened cruise among the islands in the Pacific, we perceive that the epidemic range of beriberi may be regarded as encircling the globe between the limits of the Japan Islands 37° N., and of Sydney, 33° 59' S.

In a highly valuable section on beriberi which forms part of his ‘Statistical Notes on some Indian Diseases,’ Dr. Waring in reply to the question *Does beriberi ever occur as an epidemic?* cites statistics from the records which show that, between the years 1829 and 1838, this disease occurred in the Bellary Jail only in the years 1832, 1833, and 1834. In the first of these years there was only one admission; in the second, 89 admissions with three deaths. There appears to be a misprint of figures for the year 1834; but there seem to have been many admissions and 25 fatal cases. At the Cuddalpah Jail there were, in 1833, three admissions, all fatal, and in 1834, twenty-five cases, ten of which were fatal. In these years, 1833 and 1834, there was terrible famine from failure of rainfall, over the whole of Southern India. Still, ‘in no other jail in Madras, hitherto exempt from its ravages, did this disease make its appearance.’ So also it is shown that, while the Madras 42nd Regiment N.I. was serving in China in 1845–47, 52 cases of beriberi, with three deaths, occurred in 1847, no cases having appeared in the three preceding years.

Beriberi is most common among natives, and it attacks half-castes. European soldiers have often suffered; their officers occasionally, but

far less frequently. Generally, it has been observed to spare neither age nor sex, but there are some marked exceptions to this rule. Christie,¹ writing from experience of the disease in Ceylon, says: 'I have never met with an instance of this complaint in a woman, an officer, or a boy under twenty.' When we come to notice the Brazilian disease we shall find that, there, while women, especially the parturient, are attacked in considerable numbers, 'the young of both sexes, under fifteen, and the old over sixty-five seem to be exempt.' During the prevalence of beriberi in the native prison at Singapore in 1880, 'the native female prisoners enjoyed perfect immunity.'²

Hitherto, the scientific definition of beriberi has always appeared to be one of the most obscure problems in ætiology. Probably feeling this, the College of Physicians placed beriberi, in their nomenclature of 1868, next to general dropsy, but refrained from giving it any definition. Although its manifestations are numerous and complex, I think that, on carefully studying its phenomena, we cannot fail to arrive at the conclusion that true acute epidemic beriberi is not, as some have thought, a group of diseases, but a malady quite distinct from all others—a fever of varying type, but characterised by a very marked individuality. According to their opportunities of observing it, and to the type of disease with which each authority has been most familiar, authors have assigned various causes to this malady. Thus (1) Ranking held that it is renal anasarca; (2) Malcolmson viewed it mainly in its paraplegic and scorbutic aspects; (3) Morehead considered it to be an expression of scorbutic cachexia; while (4) others have regarded it as an outcome of paludal cachexia. Each of the leading authorities on the subject adduced very cogent facts in support of his opinion. I think, however, that an analysis of the characters of the epidemic disease which first appeared in Calcutta in 1877, and Mauritius in 1878, enables us to declare (5) that acute beriberi is an exanthematous fever, hitherto best known by its sequelæ—acute general anarsaca, and frequently but not invariably, morbus Brightii and paraplegia.

I shall review these five doctrines *seriatim*, save that I shall give precedence to the consideration of the fifth.

V. Acute Beriberi an Exanthematous Fever characterised by general dropsy, effusions into the serous sacs, and other grave sequelæ.—I have mentioned above that, in 1877, a disease having all the characters of acute beriberi made its appearance in Calcutta, and became rather extensively prevalent. No European is proved to have been affected. Eurasians and Armenians suffered. A very full account of this outbreak will be found in the 'I. M. G.,' *passim*, for 1880, as contributed by various good observers, who were not at all unani-

¹ *Edin. Med. Jour.*, vol. iii. p. 116.

² Rowell.

mous regarding the nature and origin of the malady. None but those practically well acquainted with beriberi in its homes can judge with absolute certainty whether we ought to agree with one or other of the only two Calcutta men who had seen beriberi—Dr. Coates, who had observed what was called beriberi, in the Madras regiment at Cuttack, and who held that the new Calcutta disease is not identical with beriberi, and Baboo Rammay Roy, who had recently treated some 500 cases of veritable beriberi in the Madras district of Coimbatore, and had, since his return, seen some 150 cases of the new disease in and around Calcutta, who asserted that ‘they were identically the same malady.’ My own conviction, arrived at after a very full survey of the whole question, clearly is that the Calcutta disease was true acute beriberi.

The following passage will be found at page 1114 of Professor Aitken’s noble work on medicine, under ‘Beriberi.’ ‘Febrile symptoms are associated with this acute anasarca. The skin is hot and dry, the urine is scanty and high-coloured, the bowels are costive, and the stomach is irritable. There are rapid and full pulsations of the large arteries, while the pulse may be variable at the extremities, accompanied generally with dyspnoea and symptoms of effusion within the chest. In other cases there may be headache, restlessness, and delirium, with a slow and full pulse, indicating serous effusion and pressure on the brain. In the chronic form of beriberi the patient is very often more or less worn out by some previous disease, or he may have had a previous acute attack.’

In early accounts of beriberi, we frequently meet with such entries as these: ‘The disease commenced with fever, followed by swelling and numbness of the feet and legs.’ ‘Had fever last night, skin dry, pulse excited.’ In the following quotation, the italics are mine: ‘In detailing cases of beriberi, the cause supposed or probable should be first given, stating whether it was preceded by, or followed any epidemic visitation of disease in the district or neighbourhood, such as cholera, *fever*, or dysentery; and also distinguishing particularly the *acute form of beriberi, attended with increased vascular excitement*, and coming on as a primary affection, from the chronic form which is frequently the sequela of rheumatism, *fevers*, &c.’¹

‘Beriberi occurs both in an acute and chronic form; the acute form being usually attended by fever of an intermittent type, and of four or five days’ duration, whereas the chronic form is more frequently the sequela of rheumatism or of fever, either of the intermittent or remittent form.’²

In an abstract given by Malcolmson of nineteen cases of men,

¹ Editorial remarks, *Madras Medical Journal*, p. 472, vol. i. for 1839.

² *Madras Medical Topographical Report*, Northern Division, p. 90.

admitted with various diseases, who subsequently became the subjects of beriberi, five were admitted for 'fever,' and the eleven who were admitted for the four diseases—phlogosis, two; rheumatism, five; diarrhœa, one; anasarca, three—probably had commencing beriberi when they went into hospital. Malcolmson, and many who followed him, threw over this disease a darkness which has never yet been dispelled, by commencing their description of its symptoms at the beginning of the end. I cite Malcolmson's words (the italics are mine): 'It (beriberi) usually *commences* gradually with a feeling of numbness, sense of weight and slight weakness and stiffness below the middle of the thighs.' We might as well attempt to describe splenic cachexia by saying, 'It usually *commences* with a pain and sense of weight in the left side.' These observers fell into the great error of not inquiring strictly into the preceding fever. Doubtless their patients, not being properly questioned, did not enlighten them on this material point. Natives pay little attention to a single attack of fever, but usually seek relief when œdema and still graver symptoms occur. The severity of fever varies greatly in the exanthemata, and so does the amount and character of the exanthem, especially in the dark races. Judging by the light which the recent outbreaks in Bengal and Mauritius afford us, I am confident that Malcolmson, and others, erred in overlooking the fever and its exanthem, and by regarding the complications as the disease. In the epidemic which made its appearance in Calcutta in 1877, the initial fever and its exanthem were frequently overlooked, but it was generally noticed that the attack was ushered in by febrile symptoms of a few days' duration. It must be borne in mind that a malarious taint is to be suspected in the constitution of every Calcutta native; but it is clear that this fever was not the common paludal fever of Bengal. Many observers stated that there was no periodicity. Elsewhere we read of evening exacerbations and morning remissions. In February 1880, Dr. McLeod said that 'fever sometimes preceded and sometimes succeeded the œdema, and sometimes did not occur at all. It seemed to be of quotidian type when present, and well-marked rigors ushered in the attacks.'

Being a general practitioner, Dr. Chambers had an advantage over nearly all of those who observed the disease, in seeing cases from the commencement; while they, being consultants, were doubtless, in most instances, called in late. Indeed, Dr. Harvey remarked that, although he had seen many cases, he had not been able to have any one case under observation throughout. Dr. Chambers is the only reporter who says much of the cerebral conditions. He noticed marked head symptoms. He speaks of 'intense frontal headache,' and insomnia, and says that 'when the disease is ushered in with great severity, there is a tendency to stupor and wandering delirium, generally confined to one particular subject. The patient is, however,

rarely insensible, but is always roused up from the stupor and delirium by being loudly spoken to; sometimes furious delirium and violence occur.'

Frequent mention is made in the reports of an exanthem. Thus, in a girl expressly stated to have escaped fever, Tumeez Khan says, 'The limb swellings were stated to have been preceded by intolerable itching of the whole body, and the subsequent appearance of a rash all over it, and which, from the very vague description given, I think to have been of an urticarious kind.' In 1879, Dr. Juggobundhoo Bose had two cases in which 'a redness of the skin, like scarlatina, preceded the swelling.' In February 1880, Dr. McLeod noticed, from large experience, that 'the skin had been carefully examined, and in only one case was any abnormality found. This was a claret-coloured discolouration of the legs, disappearing on pressure.' But he also stated, doubtless from the patients' own imperfect accounts of what had occurred to them, that the œdema 'was preceded by burning and painful sensations, which seemed to be confined to the skin and soft parts.' In a case of Dr. Nicholson's 'there were purpuric spots.' In March 1880, Dr. Chambers, in describing the leading symptoms, mentions a 'sensation of burning heat in the body, as though the skin were rubbed over with ground chillies, preceded or followed by repeated sensations of chill; a scarlet efflorescence from the beginning of the disease, especially confined to the face, neck, upper part of the thorax, and the extremities, or petechial spots with circumscribed measly eruptions on face or trunk, or large purpural or mulberry patches. In infants a marbly, mottled, purplish or red tinge of the skin, especially of the extremities, neck and cheeks; a flushed puffy countenance, with suffused glistening eyes.' These observations become very noteworthy when we are told by Dr. Lovell that, in 1878-79, the Mauritius epidemic of 'acute anæmic dropsy,' as it was termed, was ushered in by 'slight fever, and in most cases by a rubeolar skin eruption, disappearing under pressure, sometimes ending in petechiæ or phlyctænæ and desquamation.' In Calcutta, the fever was succeeded by general anasarca. The œdema was by some described as being 'hard and brawny.' It usually commenced in the lower limbs, sometimes in the hands, spreading more or less to the trunk and head. 'œdema,' said Dr. McLeod, 'was doubtless the most constant symptom.' 'The œdema,' said Dr. Harvey, 'is general, not only throughout the connective tissue of the muscles, but the connective tissue of solid and visceral organs, in every cavity of the body, is bathed in fluid.'

Dr. Harvey noticed that 'in two cases there was an appearance of paralysis of the lower limbs, as if from effusion of serum into the spinal canal; and, in a good many more, there has been great difficulty in walking, apparently not due to paralysis, but to the local condition of the limbs.'

Tumeez Khan's expression, that, in one of his cases, there was 'inability of locomotion owing to excessive tension and stiffness of the lower extremities,' appears to represent correctly the symptom in question in many cases. Dr. Chambers notices the occurrence of epistaxis in several instances. Capillary bronchitis was frequently present; pneumonic consolidation in some cases. Death commonly resulted from large, but apparently not inflammatory, effusions of clear serum into the pleuræ and pericardium. Consequently, there was palpitation and great præcordial distress and orthopnoea. Anæmic murmurs were frequently present. Dr. McLeod, speaking from large observation, stated that 'death where it occurred would seem always to be sudden and associated with dyspnœa.' Many early writers described the suddenness of death in beriberi. Malcolmson and McLeod use nearly the same words. The former wrote: 'There is oppression and weight at præcordia, dyspnœa on slight exertion, diffused and irregular pulsation in the cardiac region.' . . . 'The patient is often found dead in bed, or sinks after several fainting fits or throbbings of the heart.' The urine was often noticed to be high-coloured, scanty (not always), almost suppressed, but there was no albuminuria or other evidence of Bright's disease. In a solitary case, which I think goes for nothing, there was 'a small quantity of albumen.' In some cases the urine was of very low specific gravity. Here and there a case is reported to have commenced or terminated with diarrhœa, or there was diarrhœa at the height of the disease, or dysentery later on; but, in most cases, there was constipation; in several, agonising pain in the lower bowel in defæcation. In many cases, in persons who were not sailors, the state of the gums gave evidence of scorbutus. In some the gums were healthy. In examining a considerable number of Bengalis, we may always expect to find that some are scorbutic, but it does not appear to be shown that scurvy was generally present among these patients. Still, Dr. McLeod observed that 'in a few cases the gums were rather swollen, and bled when the teeth were cleaned.' 'Neither an anæmic nor scorbutic condition appeared to be an essential precursor of the malady, though a certain degree of both dyscrasiæ seemed to be developed in its progress.'

Most of those who observed the disease at its outbreak, in 1877, met with many cases which did well. In 1880 there were numerous deaths. In February Dr. Cayley reported four deaths, and two persons in a dangerous condition in one family. In that month, Dr. McLeod, Health Officer of Calcutta, said that the town returns showed 266 cases and 51 deaths, and the suburban 364 cases and 163 deaths. It was observed by Dr. Harvey and others that the disease was not confined to the poor and ill-fed; rich and well-to-do people, well fed and living in well raised and very dry houses, were quite as freely attacked as the poor, living in damp huts on the ground level. 'The

disease,' says Dr. McLeod, 'lasted for two or three months in well-developed cases.' In the huge mass of evidence which I have analysed I see no mention of the duration of very rapidly fatal cases, save in one, by Dr. Chambers, in which death appears to have occurred in three days.

There are only two full reports of autopsies, made in February 1880 by Tumeez Khan. The most remarkable appearances were general œdema. Integuments of the lower limbs 'hard and brawny,' not pitting on pressure, and loaded with effused sero-sanguinolent fluid. There do not appear to have been any head symptoms during life, and no marked cerebral lesions were discovered. Large effusions of limpid serum, without traces of inflammation, in the pleural and pericardial sacs. Considerable œdema and carnification of the lungs. Heart flabby, normal in size,—(in one or both cases?), fatty. No peritonæal effusion, no noteworthy renal lesion. In both of Tumeez Khan's cases, as in most of the other reported autopsies, there was marked hepatic congestion. Blood thin and watery, with a marked excess of white corpuscles.

Much was said about the contagiousness of this disease, but no evidence of contagion is given, beyond the frequent occurrence of several cases in one family—which, standing quite alone, is of course no valid proof of the agency of contagion—and one very suspicious case mentioned by Dr. Harvey. Dr. Chambers, who was one of my most esteemed pupils and house physicians, considered that this 'new disease' was relapsing fever. He does not appear to have found support from any other observer. He shows that a tendency to relapse marked several of his cases; but I cannot think that his patients suffered from true relapsing fever. I think that he saw an exanthematous fever; but he appears to place his fever cases apart from those which, he considered, resembled beriberi.

This epidemic was observed at Dacca and Sylhet, and at Shillong in the Kasia hills.

There does not seem to have been any appearance of the disease in Calcutta after the hot weather of 1880.

Between November 1878 and June of the following year, a similar epidemic prevailed in *Mauritius*. It was stated to have caused 729 deaths. Dr. Lovell, the chief medical officer, sent a report on this disease, which he termed 'acute anæmic dropsy,' to the Calcutta Medical Society, in July 1880. I have already spoken of the exanthem which generally characterised this malady. In the worst cases, there were hydropericardium, hydrothorax, and ascites. Signs of scorbutus are not mentioned. 'Europeans were entirely exempt.' 'The patients, generally Indians, were very anæmic, the blood watery, with marked diminution of the red blood cells, and increase of leucocytes and granules.' 'The urine was seldom albuminous.' Enlargement of the spleen was not found to be a feature of the disease.

Opinions of medical men were divided regarding the contagiousness of this malady; the majority were opposed to the view of contagion.

A report which appeared in the first volume of the 'Madras Quarterly Medical Journal,' page 70, shows that, late in the year 1829, beriberi, which had previously been unknown in that locality, attacked the Madras Sepoys at Singapore, while 'the convicts remained without a single patient with the complaint.' True beriberi prevailed in the criminal prison at *Singapore* in 1875-76-78-79 and 80. It did not spread among the community at large. No European or native female prisoner suffered. In 1878, the death-rate was 16·20 per cent. of admissions, and rose to 20·63 in 1879. There is no mention of fever or eruption. Rheumatic pains and paraplegia were frequent symptoms. The great serous cavities, peritonæum, pleuræ, pericardium, were filled with serum. Scorbutic symptoms appeared in some cases. The urine was scanty, high-coloured, and sometimes albuminous. In a leading article in the 'I. M. G.' for September 1880, Dr. McLeod observed that, 'as regards symptoms and mortality, the Calcutta disease presented a sort of transition between the pure beriberi of Singapore, with rheumatic and paralytic phenomena and albuminous urine, and the acute dropsy of Mauritius, which presented many of the features of a contagious exanthem.'

I must repeat that my own inference from the evidence is, that the 'new disease,' as it occurred in Bengal and Mauritius, was an exanthematous fever. Its mode of invasion, when epidemic, would alone declare beriberi to be a fever. No one who has studied the natural history of fever will believe that the epidemic disease which, as we have seen, invaded Lower Bengal in 1877, and Mauritius in the following year, was either paraplegia or scorbutus, or consider that it was renal anasarca.

Waring says, citing from Malcolmson twenty-four undoubted cases of beriberi, in which the post-mortem appearances are given, 'the kidneys in most cases were affected, being generally found enlarged, softened, and occasionally anæmic. In four they are described as normal.'

I. Beriberi regarded as Renal Anasarca.—In a valuable but very rare volume,¹ 'The Proceedings of the Hyderabad Medical and Physical Society,' Mr. J. L. Ranking published, in 1853, a series of reports, tending to show that the previously obscure disease, beriberi, depended mainly upon the existence of a form of *morbus Brightii*. Mr. Ranking said that his limited experience led him to adopt the opinion that beriberi is primarily and essentially a renal disease; that deterioration of the blood is caused by lesion of the kidney, and that the 'numbness,'

¹ I only know of one copy, possessed by my friend Dr. Waring, C.I.E. I gave extracts from its contents in the 'Bibliographical Record,' attached to No. 2 of the *I. A.* in 1854.

staggering gait, or more confirmed paralysis of motion (pointing to affection of the spinal cord), the 'irritable' condition of the heart, the endo- or pericardial 'inflammation' (?) and effusion (which latter is generally the immediate cause of death) are secondary affections. He showed that, in sixteen cases of beriberi, the urine was more or less albuminous in eleven; in eight, the microscopic elements characteristic of kidney disease existed. Microscopical examination was made in these eight cases only; so that, in every case of well-marked beriberi which fell under Mr. Ranking's notice, the microscopic evidences of kidney disease were discovered when sought for. In nine, there was general anasarca; in one, the anasarca existed with ascites; in two, there was delirium; in one, coma; in fourteen, numbness without actual anæsthesia; in two, impaired sensibility with numbness; in six, there was more or less loss of motor power; in seven, symptoms of pericardial and endocardial affection; in five, signs of œdema of the lungs; in two, hepatitis; in two, nausea and vomiting; in two, diarrhœa. It was noticed by Malcolmson that there were certain points of resemblance between beriberi and *morbus Brightii*. This resemblance forced itself upon Mr. Ranking's attention before he had access to Mr. Malcolmson's work. Upon commencing the examination of the urine of all beriberi patients, it was found that, although the quantity excreted, the reaction, specific gravity, colour, &c., differed according to the stage and form of the affection, the microscope revealed the presence of blood-discs and epithelial scales. In two cases there were also tube casts, and mucous and organic globules. The urine was often coagulable by heat and nitric acid, though never to a high degree albuminous. He represented that, in the earlier stages of beriberi, the state of the urine points only to simple congestion or sub-acute inflammation of the kidneys. He found it to have a deep red colour, owing partly to the admixture of blood, partly to concentration; a specific gravity at or somewhat above the natural standard. A deposit of lithic acid and lithate of ammonia sometimes occurred, and abundant and large crystals of lithic acid. These morbid conditions of the urine will, Mr. Ranking considered, be found to precede the anasarca, numbness, and other symptoms of spinal affection. Unless the state of renal congestion, or sub-acute inflammation, upon which it depends, can be overcome, the anasarca continues, effusion 'into the spinal cord' (?) takes place, the heart becomes involved, and effusion into the pericardium and pulmonary œdema carry off the patient. The urine remains scanty, in such cases, to the end, or is even totally suppressed. In more chronic cases, or in individuals who have struggled through the acute stage, the urine was found to alter in character—it increased in quantity, but decreased in density—it seldom, however, reached the healthy standard even as to quantity—it acquired a pale opalescent colour—had an alkaline reaction, a

specific gravity of 1,010, and coagulated more or less by nitric acid and heat. 'Here,' said the author, 'we have all the characters of the urine of "Bright's kidney."' 'The alkaline reaction is due doubtless to the existing lesion of the spinal cord which, although in itself a secondary affection, reacts upon the urinary organs, the mucous membrane of the bladder especially, causing an increased secretion of mucus, or even pus, and consequent alkalinity of the urine. The microscope still reveals blood-discs; a great abundance of epithelial scales with mucus or blood globules entangling crystals of triple phosphate, or, if any deposit occurs, it is made up of mono-basic or bi-basic phosphates, amorphous phosphate of lime, and mucus.'

Others, who observed beriberi nearly at the same time, noticed the renal complication.

In the middle of August 1852, (dates are self-evidently of great importance in the correlation of cases of beriberi, as showing agreement or difference in type in the same or in other epidemics of the disease,) Dr. George Mackay¹ admitted to hospital, at Rangoon, a Madras sepoy, who died, in about twenty-six days after sickening, with typical symptoms of beriberi. 'The urine presented a copious deposit of mucus, with altered blood-discs and crystals of the triple phosphate.' Many other cases of a nature similar to this came under Dr. Mackay's notice during the same (second Burmese) war, but none in which the symptoms so nearly resembled those of beriberi. His attention in those cases was early directed to the state of the urinary organs. 'Epithelial scales, altered blood-discs, crystals of the triple phosphate being almost invariably met with; in some cases oxalate of lime crystals, and in one or two tubular casts were observed.'

The 'Hyderabad Proceedings,' in which Mr. Ranking's paper appeared, also published a case by Mr. Maillardet. Dr. Van Someren also discovered 'casts of uriniferous tubes, epithelial scales, altered blood-discs, and octohedral crystals of the oxalate of lime.' We have here descriptions, by four observers, of the conditions of the urine in (epidemic?) beriberi, as the disease presented itself within a short space of time—apparently a period of a few months—on the continents of Madras and Burmah. Although the above facts, being published in the 'Indian Annals of Medical Science,' must have come before many observers in India, I do not find that Ranking, Van Someren, Mackay, or any one else, up to 1875, recorded other cases of albuminuria, cast desquamation, &c., in beriberi. Was this because, during that particular epidemic and that one only, the kidneys suffered nearly as they do in the cognate but very distinct (from epidemic acute beriberi) exanthematous fever scarlatina? At any rate, regarding beriberi as a fever of this genus, I am quite prepared to find that, when the exanthem is checked by the 'chill,' so frequently mentioned as a

¹ *I. A.*, No. 5, p. 349.

prominent exciting cause in this disease, albuminuria may occur. It is clear, however, that albuminuria was not a marked feature in the late Bengal and Mauritius epidemic (it is stated that, in the latter outbreak, 'the urine was seldom albuminous'), although it is to be regretted that none of the reports which I have seen take any notice of the microscopic condition of the urine.

It will have been noticed that, in the Singapore outbreak, which commenced in 1875, 'the urine was scanty, high-coloured, and sometimes albuminous.' 'Fatal cases,' says the reporter,¹ 'are preceded by almost complete suppression of this secretion. The kidneys have been found to be pale, enlarged, and fatty.' Aitken says, not apparently with reference to this particular outbreak of beriberi, that 'as the disease progresses a total suppression of the urine generally takes place.'

Undoubtedly the incidence of beriberi is especially directed to those who are the subjects of renal disease. It has always been observed that a very considerable proportion of the victims of beriberi are drunkards. Intemperate European soldiers are liable to this malady; their officers are far less so, although they have not always escaped. Christie says in his report on beriberi in Ceylon: 'I have remarked that a very great proportion of the patients seized with this disease were men accustomed to lead a sedentary and debauched life, such as tailors, shoemakers, &c., who, when working at their trade, are often excused the duty of the field, and by their double earnings are enabled to procure a larger quantity of spirits than the other men.' I was told at Aden, about twelve years ago, that European soldiers posted there, who are temperate, generally escape; but that, when a man becomes a sergeant, he is not unlikely to take to free living, and then probably is attacked with beriberi. At the same time, it is clear that, while beriberi sometimes resembles Bright's disease, it by no means always assumes that character. Certainly it is not produced by the ordinary causes of renal anasarca; and, if some of the cases of beriberi, such as those observed by Ranking, Mackay, and others, may be designated as examples of this form of general anasarca, ordinary renal anasarca is in no sense beriberi. The paralytic element of beriberi is not characteristic of common *morbus Brightii*.

II.—Beriberi viewed in its Paralytic Aspect.—One of the leading sequelæ of beriberi being anasarca and dropsical effusion into the areolar tissue and serous cavities, the occurrence of spinal paralysis, in various degrees, as one of its complications, is clearly accounted for. As I have shown, in describing the Bengal epidemic, the stiffness of the lower limbs is frequently due to the presence of hard cedema, not to spinal paralysis; but, in many cases of beriberi, distinct

¹ I cite here the report by Dr. T. Irvine Rowell, P.M.O., Straits Settlements, for which I am indebted to the kindness of my friend, Dr. Waring.

impairment of motor and sensory power in the lower limbs has been observed.

In an abstract of Malcolmson's prize essay on beriberi, Sir G. Ballingall says:— 'In dissecting patients who have died of this disease, serous effusions, more or less extensive, into the cellular substance, as well as into the great cavities, would seem to be the more prominent appearances; and, in addition to this serous effusion, there often exist in the lower region of the spine, about the origin of the lumbar and sacral nerves, symptoms of congestion; and, in one very remarkable case, of which the details are given by Mr. Malcolmson with an illustrative engraving, an effusion of reddish coagulable lymph had taken place on the posterior surface of the theca, at the fourth dorsal vertebra, and the same in the region of the sacrum.' In his report on beriberi, Waring says (citing Malcolmson): 'The spinal cord, in one instance, presented almost total decay of the white matter of the nerves of the cauda equina, but the cord itself was healthy; in another there was a copious deposit of red lymph, which had become adherent to the spinal cord; in a third the cord was very vascular; in three others it is described as healthy.'

Although the form of chronic paraplegia, termed by Bontius and other old writers on the diseases of the East 'barbiers,' is, as shown by Henry Marshall, quite distinct from beriberi, it can hardly be doubted that much of that which was formerly regarded as barbiers was, in reality, beriberi with spinal paralysis.

Lathyrism, paralysis of the lower limbs caused by habitual feeding upon the *dāl* of the *Lathyrus sativus*, prevails extensively in Upper and Central India, especially near Allahabad and in Upper Scinde. M. Proust has observed it in the Jurjura mountains of Algeria, and it is stated to be far from uncommon in France. A recent writer having stated that lathyrism and beriberi are identical diseases, M. Pierre Marie¹ has undertaken to prove that such is not the case. It appears to me not improbable that the writers have confused beriberi with barbiers. At p. 113 of the 'Indian Annals of Medical Science,' No. 23, Dr. James Irving has contrasted the symptoms and other conditions of lathyrism and barbiers. I should have considered beriberi and lathyrism as little congeneric as Monmouth and Macedon—beriberi being a very acute disease, lathyrism, as it occurs in India, a chronic one. But, since the question has arisen, M. Marie's paper calls for notice from medical men working in those districts, far separated as they are, in which lathyrism and beriberi are most common; lathyrism prevailing far inland, beriberi occurring almost entirely within the influence of the sea breeze. It appears singular that, in giving long and minute descriptions of the conditions of the

¹ *Progrès Médical*, No. 43. An abstract of this paper is given at p. 606, *M. T.*, vol. ii. for 1883.

lower extremities in the contrasted diseases, M. Marie appears to have overlooked œdema in beriberi.

The loss of power in the lower limbs, so frequently noticed in beriberi, appears to need clear definition. In what cases is it dependent upon spinal lesion, and in what others is it due to hard œdema of the extremities and debility? With regard to the former cases, one point ought to be cleared. Sir Joseph Fayrer has suggested the probability that filarious disease may be in some way concerned in inducing beriberi.¹ A form of spinal paralysis in the horse, *kumree*, which prevails within the Madras area of beriberi, and in Eastern Bengal, has been ascribed by Twining and others² to the presence of entozoa in the cord. Do spinal lesions in beriberi ever arise from this cause?

Undoubtedly, although spinal paralysis is a recognised sequel of beriberi, it cannot be considered that beriberi is, in any sense, paraplegia.

III.—Beriberi regarded as an Expression of Scorbutic Cachexia.—Marked evidences of scorbutus are so often noticeable as prominent symptoms in cases of beriberi, that at least one of our best Indian observers has considered that beriberi is essentially a form of scurvy.

Wishing to give fair play to this, as well as to all the other leading theories upon the causation of beriberi, I may cite the following rather striking example of this coincidence, as observed by my late esteemed friend Tumeez Khan Bahadur, in Calcutta, in 1878, and others which I find in the ‘Indian Journal of Medical Science,’ vol. v. N. S., p. 75, as occurring *circa* 1839 on a passage from Java.

1839.

A Dutch barque arrived in the Madras Roads, from Java, on May 12, having left Malacca, where she lay only three or four days, all being well, on April 7. The crew were Javanese, who had made objections about coming to the Coromandel coast, as they said ‘many of their countrymen had died of a similar disease on former voyages.’

‘About twenty days before reaching Madras, many of the crew were seized with swellings and numbness of the legs and feet, but yet were able to perform their duty; when they arrived within fifty or sixty miles of the coast, and within the influence of the land winds, in three cases the symptoms became much aggravated, they were unable to walk, dyspnœa and pain in the præ-

1878.

A Mahomedan ship-captain of one of the Arab sailing vessels which ply between Calcutta and Jeddah, a native of some part of the Coromandel coast, his son, and a third member of his family, both said that they had returned hale and hearty from a trip to Jeddah, the third person had died previous to Tumeez Khan’s visit.

A short time after their arrival in Calcutta ‘both were nearly simultaneously attacked with febrile symptoms, which lasted for a few days, and were followed by œdema of the lower limbs, to which succeeded cough and dyspnœa’ (the son had hæmoptysis). The ‘œdema extended up to the loins.’ When visited, they had ‘orthopnœa, palpitation, and inability to lie down.’ It is not said

¹ Quain’s Dictionary, p. 105.

² Vide *C. M. T.*, vol. i.

1839—*cont.*

cordia supervened, and they died the day after the ship arrived.

‘They were a wretched-looking set, and were very badly fed, their rations consisting only of rice, and of that a very limited quantity. The water was bad, and they were on very short allowance.’

1878—*cont.*

whether the father and son died, but they probably did.

‘Both had spongy and discoloured gums.’

In connection with the above observations, all who are desirous to investigate the nature of beriberi ought to study Morehead’s chapter on this disease. His data are mainly derived from histories of ‘beriberi as it occurred among the Lascar crew of the ship “Faize Allum,” in May 1853, when in lat. 10° north, within sixteen days’ sail of Bombay, and from an account of an outbreak of “epidemic ascites,” or “peritonæal dropsy,” in thirty of the crew of H.M.S. “Juno,” on her arrival at Sydney from a lengthened cruise among the islands of the Pacific, in January 1856.’

The native subjects, four cases of beriberi, from the ship ‘Faize Allum,’ whom Morehead treated, were doubtless scorbutic; the men of the ‘Juno’ were probably so. Morehead does not appear to have met with any other cases of beriberi. He considered that ‘beriberi, more particularly in its acute form, occurs usually in persons favourably circumstanced for the development of a scorbutic taint, and subsequently exposed to cold, dry, or moist winds, or to lying on the ground wet with rain or dew.’ With the judicious circumspection, which characterises all his reasoning, he observed that to him ‘it seems that beriberi is a general dropsy, and that, in regard to each instance, the question ought to be—what is the pathology of this case of general dropsy?’ He added, ‘generally it will be found that a scorbutic diathesis and external cold or wet, are the determining conditions.’¹ Dr. Peet adopts this view without qualification. He says, ‘When individuals who are scorbutic are exposed to cold and alternations of temperature, they are not unfrequently attacked with general dropsy. It has been customary to designate the disease now referred to as beriberi.’² Here is, I think, another example of the common error of regarding as cause and effect two circumstances which are merely associated as frequent coincidences. It is to be borne in mind that beriberi is often clearly epidemic, the epidemicity of scurvy being a questionable point. There was no beriberi among the scorbutic mariners of last century; and, in many cases of sea scurvy which I have treated, there was not the very slightest evidence of its existence. Beriberi occurs in some localities notorious for scurvy, as Aden; but it

¹ *Op. cit.* pp. 707–8.

² *Principles and Practice of Medicine*, p. 570.

does not prevail in certain of those districts of Hindustan—as the province of Behar, and in Scinde—in which scurvy is extremely prevalent. Scorbutus, as we have already seen, occurs in Lower Bengal, but not so extensively as in some other Indian districts. The recent Bengal epidemic frequently attacked the scorbutic, but it was noticed that many of the sufferers from the epidemic were free from evidences of scorbutus. I think that, when we state the fact that, wherever beriberi prevails, it especially selects the scurried, we are free from the error of supposing that acute beriberi is an expression of scorbutic cachexia, although scorbutus should be looked for in all cases of beriberi, and be treated, when present, as a grave complication.

IV.—Is Beriberi, as some have considered, a Form of Malarious Cachexia?—Although it can hardly be doubted that, especially in old times, many cases of malarious cachexia have been registered as beriberi, I think that this question must be answered in the negative. Beriberi prevails in localities, such as Aden and Mauritius, where the paludal poison does not abound in great intensity. Until 1877, the delta of the Ganges, that hot-bed of marsh influence, was never visited by this pest as an epidemic, and has never generated endemic beriberi. I believe that, up to the time of my leaving Bengal, ten years ago, it was never suggested that beriberi could originate there. Considering that a vast number of sailors pass through the Calcutta hospitals, I think it noteworthy that only one not unquestionable case of this disease, in a half-caste sea-faring man, made its appearance in my wards. No one acquainted with the natural history of the marsh poison would, in studying the outbreaks of beriberi in Calcutta and Mauritius in 1877 and the following year, consider that there could have been any interchange of paludal influence between these two widely separated localities. Nevertheless, seeing how generally and how often gravely prevalent malarious cachexia is in most of the regions which beriberi visits, the necessity of searching for it, as a most serious complication, must never be overlooked. Few constitutional states can be regarded as more perilous than that of the multitudes of unfortunates who, being the subjects of the combined cachexiæ of marsh-poison and scorbutus, are attacked with beriberi.

I think that we are justified in concluding that beriberi is not a malarial fever, but that the victims of malarious cachexia are especially liable to suffer from its attacks.

Treatment.—If I am right in the firm belief which I hold that true beriberi is an exanthematous fever which, although wholly *sui generis*, pursues a course which, in more than one of its leading actions, much resembles that of scarlatina, it is evident that here our first object should be, as it is in scarlatina, to endeavour to see each case *ab initio*, and to prevent the occurrence of renal and other visceral mischief, anasarca and dropsical effusions into the serous

cavities. If it could be my duty to have charge of military and jail hospitals within the endemic area of beriberi, every European soldier, sepoy, and prisoner would be warned to seek relief from my assistants *immediately upon being attacked with even the very slightest symptom of fever*. I would myself examine all fever cases early, within twelve hours if possible, carefully searching for the exanthem on the lighter skins, and considering that, if I frequently discovered it there, its presence might be suspected in those of darker complexion. Then the chief indications would be:—To admit all fever cases as in-patients; to avoid the occurrence of that CHILL so frequently assigned as the cause of general anasarca in beriberi; to promote healthy cutaneous action and to subdue fever; to discover and deal with pre-existing renal and other visceral disease, and with malarial and scorbutic cachexia. After remaining for two or three days under observation, the cases of ordinary intermittent fever would explain themselves, and would then demand less close attention. When patients do not seek relief until general anasarca has become established and, being too weak to go about, are desirous to come to the hospital as a refuge, the same principles of treatment ought to guide us. With the deepest respect for the memory of a great Indian physician, I cannot agree in the validity of his maxim, guarded as it is, ‘remove the dropsy by purgatives or diuretics, being guided to the use of the one or the other by the state of the pulse.’ I would no more give direct diuretics in the anasarca of beriberi than I would in that of scarlatina. My great effort would be to obviate renal congestion in all possible ways, and to make the work of the kidneys as easy as possible. I would not give anything more drastic than the mildest laxatives in these cases, liable as they are to end fatally by diarrhoea or dysentery. If we can only remove the other complications of beriberi, the anasarca will undoubtedly disappear as constitutional power becomes re-established. No purgative or diuretic will, except by promoting exhaustion, touch a pericardial, arachnoid, or pleural effusion which threatens life.

I believe that, fifty years hence, the futile and destructive practice of endeavouring to remove dropsical effusions by the use of drastics and direct diuretics will be viewed as one of the most barbarous therapeutic errors of the present century.

I think that the whole subject of acute and chronic beriberi, especially as they occur in the Madras Presidency and in Ceylon, ought to be thoroughly reinvestigated by sound pathologists. Work out the subject of beriberi, is one of the best pieces of advice that can be given to a young physician entering upon a career in the Madras Presidency.

The field of observation is considerable in Madras. The returns show that among European troops only one case occurred in the first of the four years 1880–83. Among the sepoys the admissions and

deaths were 142 with 19 deaths, 102 with 20 deaths, 141 with 13 deaths, 64 with 17 deaths. Prisoners 10 with 3 deaths, 118 with 50 deaths, 3 with no deaths, and 1 admission not fatal.

Especial care should be taken to ascertain the precise nature of what is termed the 'chronic' form of beriberi. Are these cases of malarious and scorbutic cachexia not allied to true beriberi, or are they examples of repeated attacks of beriberi and consequent ill-health? Christie observed that, 'men who have once had the complaint are the most subject to it in future.' In the epidemic and acute form, light is needed with regard to the precise type of the fever, the character of the exanthem, the range of temperature, the chemical and microscopical conditions of the urine, &c. All observers have noticed that, although the œdema of the lower limbs is hard, the serous effusions, arachnoid, pericardial, pleural, and peritonæal, the pressure of which tends to cause death in beriberi, are distinctly *non-inflammatory*. A little sifting would clear this question, enabling us to judge whether we ought or ought not to draw off the fluid from the pleuræ and pericardium, and with what prospect of success.

Note on Kakké, the Beriberi of Japan.—I am indebted to the kindness of Surgeon-General C. A. Gordon, for a sight of Dr. Duane B. Simmons's elaborate report.¹ The author defines beriberi as a disease occurring during the summer months, especially in the seaport towns on the eastern and southern coasts of the Japanese islands, chronic as to form, but subject to exacerbations of varying degrees of severity, having for its characteristic symptoms anæsthesia of the skin, hyperæsthesia and paralysis of the muscles, anasarca, palpitation, cardiac and arterial murmurs (in the wet form), præcordial oppression, abdominal pulsation. It has two distinct forms, the wet (*beriberia hydrops*) and the dry (*beriberia atrophica*); cases of both are met with in the same locality, the former more frequently when the summers are attended by an unusual amount of rainfall and moisture, and the latter when the season is of unusual dryness. The wet form is by far most to be dreaded, as it frequently runs a rapidly fatal course in defiance of all known modes of treatment. The dry form is rarely fatal.

Dr. Simmons's report will, I think, in the future, be most useful to those who may undertake the investigation of this disease in its more chronic endemic forms. He does not appear to recognise the malady, in its epidemic form, as occurring in Japan. In his histories, an early stage of fever does not appear to have been at all generally looked for; but Case I. has the following early history:—*Pernicious Beriberi; Death.*—'Entered the hospital, June 15. During the first days of May, began to feel unwell, had occasional slight chills, followed by heat flashes [flushes?]; 'a general sense of *malaise* and a tired feeling

¹ *China Imperial Maritime Customs, II. Special Series, No. 2. Medical Reports for the half-year ended March 31, 1880, 19th issue, p. 38.*

in the legs.' The following remark appears to show that Kakké does commence as fever. Dr. Simmons observes (p. 68) that, notwithstanding the strong resemblance which exists between marsh malarial fever and beriberi, their admixture is uncommon. 'They have a certain resemblance in their initial stages which is very puzzling, and, though we may be able to diagnose with certainty beriberi or malarial poison as the cause of the indisposition, nothing but the administration of quinine will, for some days, enable us to decide which it is.' 'Upon this, I must state the platitude that this or any other disease which, for days, runs such a course that the QUININE TEST alone enables us to differentiate it from 'Marsh Malarial Fever,' must be, unquestionably, *a fever*. The author continues: 'The almost invariable low temperature, in uncomplicated cases, is commonly noticed by all recent observers. So well established is this point that it is of decided diagnostic value in doubtful cases.' There is no mention of an exanthem, but I apprehend that in those cases where patients with beriberi are 'attacked with typical typhoid fever,' the eruption must not be always wanting. 'In the wet form of the disease, the urine is always less in quantity than normal. It is rarely high-coloured and is without albumen.' I think that a full and patient inquiry into the febrile stage of Kakké is still most desirable. Dr. Walter Dickson observes that, 'recent researches in the ancient medical literature of China and Japan show that the disease was known and described 2,000 years ago, and the two great varieties of dropsical and paralytic were recognised and differentiated in Japan so far back as the tenth century.' He adds that 'at present, China does not seem to suffer from this disease; but in Japan sickness and mortality from it are still very considerable.'

Beriberi in Brazil.—Dr. Walter Dickson, R.N., has most kindly allowed me to cite, from his presidential address delivered at the Epidemiological Society, some data from the history of *Beriberi in Brazil*, published by Dr. Feris in the 'Archives de Médecine Navale' for 1882. It appears that the first incidence of this epidemic was at Bahia in 1864. Soon afterwards it extended to Para, Pernambuco, Maranhão, and other Brazilian ports, speedily extending inwards to provinces remote from the seaboard, and everywhere causing a heavy mortality. In the war with Paraguay, both the armies and fleets engaged were described as 'decimated.' The relative mortality to the number of cases has varied from 74 per cent. at its first outbreaks at Bahia, 1864–66, to 13 per cent. in the hospitals of Rio Janeiro, when after a few years, in 1869–75, its nature and treatment were better understood. The mortality among the European troops amounted to 25 per cent. of those attacked, and that among the convicts in jails was as high as 37 per cent. I infer from the narrative that there is an *acute* and a *chronic* type, the duration of the former being ordinarily from four to

eight weeks, but five days and seven months are quoted as occasional extremes, while the chronic form lasts for several months and even years, with alternate remissions and exacerbations. The symptoms mentioned are anæmia, dyspnœa, dropsical effusions and paralysis. Death often occurred with appalling suddenness from embolism or syncope. So Mr. Ridley¹ noticed the suddenness of dissolution in most of the cases which he observed among the troops in garrison at Ceylon in 1814. Very frequently, when speaking to one man, he says, 'I have been called to another, whom I had just before left under promising circumstances, and have found him gasping, his eyes protruding, his hands clenched, and in a few minutes he was dead; and it has sometimes happened that the man I was addressing has been taken off in the same manner.'² While it is stated that, in Brazil, the disease has been accounted as essentially non-contagious, yet Professor Feris and Dr. Silva Lima cite some remarkable instances which seem to be more than coincidences. Three French priests affected made a journey in the province of Minas Geraes, from Caraça to Diamantina, by way of Mariana, stopping at these places, and there, and at no other localities, the disease soon after appeared. Two women died of beriberi, and soon afterwards their husbands were stricken with the malady. Two negroes were admitted to hospital for trivial surgical disorders, occupied beds which had been used by patients with beriberi, and were both seized with that disease. Such cases are considered exceptional and inconclusive, yet the malady would seem capable of being imported, for its first appearance in the Antilles, Guiana, and Brazil was almost simultaneous with the first introduction of coolie labourers from Asia. Dr. Paterson, of Bahia, who, with Dr. Silva Lima, first recognised the disease as beriberi, wrote to Dr. Aitken that he finds it 'attack persons in the most favourable conditions of life, as regards means, residence and regularity of life.' Its favourite victims are parturient women, alike before and after confinement. According to Dr. Feris, women suffer chiefly from the paralytic and nervous symptoms. The young of both sexes under 15 and the old over 65, seem to be exempt. In school dormitories, instances often occur of youths being affected, while those a little younger, in beds side by side with them, escape.

Beriberi in British Burmah.—It is shown at p. 92 of the 'S. C. R.' for 1880 that, from July to October of that year, there was an outbreak of beriberi which killed fifty prisoners out of 118 attacked in the jail at Thayetmyo, in British Burmah. I have not succeeded in finding any clinical history of this outbreak.

It is announced that, at least up to September 1885, beriberi was committing serious ravages at *Acheen*. More than 1,500 deaths had occurred during the first half of that year. Among these were many Europeans.³

¹ Cited by Aitken.

² *Dublin Hospital Reports*, vol. ii. p. 234.

³ *I. M. G.*, January 1886.

DENGUIS.

On several occasions, but especially in the years 1824, 1853, and 1871-72, severe epidemics of dengue have traversed British India so generally that few have escaped the visitation. I have been at some pains to investigate the history of this disease in India; but I think that, for practical purposes, a bibliographical foot-note of the principal narratives will furnish sufficient data for those who desire to inquire into the origin and progress of the various epidemics.¹

The last great epidemic, which reached India late in 1871, and prevailed there far into the following year, appears to have been the most general and severest that has occurred. The disease was traced down the Red Sea to Aden,² and it extended throughout the three Presidencies of India, attacking multitudes. At one time nearly the whole of the large staff of medical men in Calcutta were more or less ill with it. Then, as in previous epidemics, the words of Kenneth Mackinnon were applicable to it: 'The disease very seldom has proved fatal, perhaps never, except in cases where, from other causes, there existed a great tendency to death.' One patient, an apparently healthy man, being attacked in sunstroke weather, appeared to die from the irritation of the exanthem, as if scalded. The 'break-bone' pains often continued for years. Four years afterwards, a man accosted me in Paddington, telling me that he had been in my ward with dengue, and was only then strong enough to return to Calcutta. I inquired of myself whether I was to blame for this slow recovery, but decided that I was not. I practised throughout the epidemic of 1853, but left India when that of 1871-72 had prevailed for three months. The urine was not albuminous, nor was there anasarca or serious throat affection in any of my cases in either epidemic. One of my colleagues, finding albuminuria in some cases in the visitation of 1872, and observing the general characteristics of the fever, argued that the disease was true scarlatina. It is probable, however, that his experience of scarlet fever was limited, and I do not think that he made any converts to his opinion. It is evident that, where the epidemic scarcely spares any-

¹ Mellis—*C. M. T.*, vol. i. (1824), p. 310. Kennedy—*Ibid.* p. 371. Twining—*Ibid.* vol. ii. p. 1. Cavell—*Ibid.* p. 32. Mouat—*Ibid.* p. 41. Henry Goodeve—*Ibid.* vol. ix. (1844) p. 142. Kenneth Mackinnon—*Diseases of India*, p. 113. Edward Goodeve—*I. A.* (1855) vol. i. p. 248. Charles—*I. M. G.*, February 1872, p. 25. Fayrer—*Tropical Dysentery, &c.* p. 314. At the London International Congress of 1881, Dr. James Christie read an account of the Dengue epidemic of 1870-75, which, he considered, originated *de novo*, after a great visitation of cholera, at Zanzibar. He gave a history of previous Dengue epidemics. Dr. Christie afterwards published this paper as a separate pamphlet.

² It prevailed so generally at Aden that it was popularly said that the word 'Dengue' was an abbreviation of '*Aden Ague*'—the initial letters being omitted.

one, the subjects of functional and organic renal disorder are not likely to be exempted; and, in many cases, the exanthem is so extremely severe that it becomes matter for surprise that albuminuria should not be set up.

Quinine, in small doses, was evidently useful as a means of supporting vitality and of abating the intensity of the fever, as it is in all Indian fevers, but it exerted no specific power in cutting short the malady; which, however, when the attack was very prolonged, was evidently arrested by change of residence—a measure which is of great validity in the treatment of many Indian diseases. A member of my own family was very ill at Howrah in 1853, and made no progress towards recovery from day to day. Having removed others with great advantage, I took my patient out of our comfortable home, across the river to a Calcutta hotel. Defervescence commenced immediately, and recovery was speedy.

TYPHUS.

I do not believe that true typhus occurs in any part of Bengal Proper, *i.e.* in the southern portion of the Bengal Presidency. In upwards of twenty-seven years' practice there, during the last fifteen of which I had sixty beds in the largest Calcutta hospital, I looked constantly for the disease, but never saw it. Still the local medical literature contains more than one statement adverse to my dictum. Thus, questionings have, from time to time, arisen as to whether the Burdwan fever, which most observers regarded as a paludal remittent of great severity, was not typhus. In 1864, Dr. Chuckerbutty received into his wards at the Calcutta Medical College Hospital twelve cases of what he concluded to be typhus fever. He showed me one or two of these cases at the time. They were certainly like cases of typhus; but no one can distinguish a severe case of paludal remittent from one of typhus by merely looking at it, without submitting it to the crucial test of treatment. He and I admitted on alternate days—our wards opened into each other—and I watched vigilantly for the occurrence of cases of true typhus in my own beds, but they never came to me. I do not think that the slight account¹ which Dr. Chuckerbutty has given of these cases could have convinced readers of large English and Indian experience that these were genuine cases of typhus. The most striking fact is that, in eleven of the twelve cases, there was a 'mulberry rash;' but petechiæ are seen in several Indian fevers. There was no evidence of contagion.

The occurrence of typhus in Upper India rests upon more extended authority; still I hold, with Drs. Morehead and Murchison, that proof is wanting of the existence of genuine typhus in any part of India.

Five cases of what he considered to be typhus have been recorded²

¹ *I. A.*, No. 18, p. 122.

² *C. M. T.*, vol. viii. part 11, p. 274.

by Dr. Allan Webb as occurring at Simla. Contagion was not proved; and, during nearly fifty years which have elapsed since these observations were made, there have been no other cases of typhus reported from Simla, the hill residence of the Government of India.

Dr. Joseph Ewart reported¹ two cases of typhus attacking native prisoners in the Ajmere Jail in 1855. The fact that their disease was considered to be typhus by an observer so accurate as my friend is assuredly strong evidence. Still I am not prepared to be convinced by two isolated cases to which the quinine test was not applied.

The late Dr. Robert Lyell and Dr. Thomas Farquhar described a fever which prevailed in 1852-53 in the Yusufzaie Valley, about forty miles north of the city of Peshawur.² Dr. Farquhar commenced by designating the malady as 'typhus fever.' The following are salient points in his description. It succeeded 'an unusual amount of ordinary intermittent or remittent fever.' 'Jaundice in most cases occurred almost from the first, and, as a principal feature of the disease, was present in by far the greater number of cases.' 'Relapses, from their almost constant occurrence, formed another particular feature of the disease.' In these relapses, 'frequent purging, great prostration of strength, and emaciation' were the most marked symptoms. 'Inflammation and suppuration of the glands in the groin, axilla, and neck occurred in some that survived the first or second relapses.' 'The relapses of the fever assumed the remittent or even intermittent type.' During these relapses, quinine, with which Dr. Farquhar was not successful at the outset of the attack, 'did a great deal of good.' With great deference to the judgment of my esteemed brother-officer, I feel convinced that this fever was very different in type from the true typhus of which I had twelve years' observation in London. Dr. Lyell seems to have approached more nearly to the true differentiation of this malady when he wrote: 'It appears to be the common remittent of the country, aggravated by filth and overcrowding into a type of fever differing very little from the relapsing fever which prevailed in Edinburgh in 1843-44.' In like manner, much questioning arose as to whether a fever which was described by Dr. W. Walker as committing great ravages in the Central Jail at Agra in 1860, which reappeared there in 1864, and which, in 1865, showed formidably in the Umballah, Futtyghur, Allyghur, Jyepore, Malwa, and other jails, was 'true typhus,' 'contagious fever,' 'typhoid fever,' 'yellow typhus,' or 'contagious jaundice fever.' Most of these reports were very carefully reviewed in the 'I. M. G.' in 1867 and 1868, by my friend Dr. David Boyes Smith, of Netley, with the result of convincing many, of whom I am one, that the Agra and Umballa fever approached very nearly in type relapsing fever, the occurrence of which in various parts of India is now scarcely denied by anyone.

¹ *I. A.*, No. 7, p. 65.

² *Ibid.*, No. 3, p. 16, and No. 4, p. 504.

If we put aside the exanthem of typhus—which, if this fever occurred in dark-skinned natives, might not be apparent—there are few things in disease which more closely resemble each other than an advanced case of Indian paludal remittent without bowel complication and a case of genuine typhus. Assuredly I know no mode of examination by which I could distinguish one from the other. But there is a test by which, in the course of an outbreak of such, at first sight, doubtful cases, I could very soon discover which of these two fevers I had to deal with. No case of relapsing, enteric, or typhus fever was ever cut short by quinine; whereas a case of paludal remittent without bowel complication, however closely it may simulate true typhus, will always, provided recovery be possible, be checked, and with more or less difficulty cured, by a well-managed course of quinine. To this QUININE TEST every obscure case of fever in India ought to be submitted by the young practitioner, who must not be too readily puzzled or deterred if it appears to fail him again and again; because he must remember that, when a patient is brought to him absolutely and irrecoverably death-stricken by a malarial fever, quinine can no more avail to put the fever out than all the fire-engines in London can save a tallow factory which has been burning for an hour.

Considering how very similar a grave case of Indian malarial remittent is to one of typhus, it is exceedingly noteworthy that, in the four years 1880–84, no case of typhus as occurring in a European soldier, child, or sepoy, appears in the S.C. returns. One case (of recovery) is reported to have occurred in a jail in 1880, and two in 1882. Here, however, an important question has arisen upon which two parties, whose opinions are equally deserving of respect, strongly differ.

Knowing that there prevailed, of late years, in northern India, a very deadly fever, regarding the type of which medical authorities were disagreed—as to whether it was malarious remittent, Indian relapsing, or true typhus, I have made inquiry of Surgeon-General De Renzy, C.B., who, while this fever was rife, held the post of Sanitary Commissioner, Punjab. Did he see true typhus up country? Dr. De Renzy informs me that his first experience of typhus in the Punjab was at Rawal Pindi, in the jail. He asked Dr. James Fairweather, who was in charge, and had had great experience of typhus in practice in Scotland, to give him notes of a number of the cases, and Dr. De Renzy had them printed in his ‘Punjab Sanitary Report’ for 1869. The list unfortunately included one or two cases of typhoid. The great bulk of the cases was of typhus. Up to that time, it had been universally believed that typhus was absent from the Punjab. Murchison referred to this epidemic in his book on Fevers. In 1872–3 there was another outbreak in the Pindi jail, Dr. John Ince being in charge. He reported that the fever was a malarious remittent, but again the mortality was so excessive that Dr. De Renzy felt

doubts as to the correctness of the diagnosis, and he moved the Government to depute a medical officer familiar with typhus to investigate the epidemic. Surgeon Lambard, who had obtained high honours in the Dublin school, was appointed, but unfortunately was attacked by the fever in a few days and died. He had no doubt whatever that the disease was typhus. Then Dr. Ince took up the record, and his notes of cases appeared in Dr. De Renzy's next sanitary report. The next epidemic that Dr. De Renzy had to deal with occurred in the Bannu jail, he thinks in 1874. This was reported as a very bad outbreak of malarious remittent; but, as the mortality was very great, and as the troops in the cantonment a few hundred yards off were quite healthy, Dr. De Renzy expressed doubt of the correctness of the diagnosis, and requested that detailed notes of cases should be kept and sent to him. Unfortunately the two medical officers who successively took charge caught the fever. Dr. Farrell was very obstinate, and would not listen to the suggestion that the fever was other than a bad form of remittent. His case was very carefully noted by Surgeon Booking, then fresh out of Stevens's Hospital, where he had seen much of typhus, and had himself suffered from the disease. These notes were published in the Appendix to Dr. De Renzy's next report. Dr. McKenzie had a very narrow escape. Dr. Booking had no doubt whatever that the outbreak was one of typhus. The last outbreak of which Dr. De Renzy speaks occurred in 1878. He had then left the Punjab, and his information is derived from notes which Dr. Johnson, who died of the disease, gave to Dr. R. Gray, who attended him in his last illness, to be sent to Dr. De Renzy. The epidemic was very fatal at Abbotabad. A young medical officer having been attacked, Dr. Johnson took him into his house, and nursed him through his long illness, but caught the infection just as his friend was convalescent. Drs. Johnson and Gray had no doubt that the disease in this case was true maculated typhus.

The Peshawur jail was, for many years impested with a most fatal fever. It was called malarious, but Dr. De Renzy is morally sure it was either typhus or relapsing, and there was good reason for believing that the Pindi jail was infected from Peshawur, and the Lahore and other jails from Pindi. Dr. De Renzy is not aware of typhus having spread *below* Lahore, or perhaps Umritsur, 'but,' he adds, 'relapsing fever flourishes in all parts of the Punjab. Epidemics of these fevers are constantly reported as remittents, and the proper measures of prevention were neglected while there was a chance of confining them within moderate limits.'

Regarding the outbreak of 1878, spoken of above, the Sanitary Commissioner says, in his Report for 1881, pp. 90-91, 'The death-rate in the Lahore Central Jail was 157·33 per mille, and in the Rawal Pindi Jail 195·33.' At Lahore Jail, 46·84 of the total mortality was due

to cholera. There was no cholera at Rawal Pindi; nevertheless a death-rate of nearly 20 per cent. was recorded, which, though a great improvement on the ratios of the previous two years, evinces a most unsatisfactory condition. In 1878 the total death-rate of this jail was only 26·35. The causes of the excessive sickness and mortality in this jail, which commenced in October 1878, and continued till April 1881, have recently formed the subject of special correspondence. ‘*Whatever may have been the exact nature of the fever which proved so fatal, and on this point there is decided difference of opinion,*’¹ there can be no question that removal from the jail was imperatively called for, and the beneficial results which followed this movement, when it was at last carried out, convey an important practical lesson which cannot be too strongly impressed on all jail authorities in India.’

We read, at page 71 of the Sanitary Commissioner’s Report for 1880, that, in reporting on the mortality in the Rawal Pindi Jail, Dr. Dallas, the Inspector-General of Jails, said that ‘for some years past the entire district, together with the adjacent districts of Hazara and Peshawur, had been suffering from typhus fever.’ Seeing, however, that, in this same report for 1880, the Sanitary Commissioner gives, for all India, one case of typhus in a prisoner who recovered, no admission for relapsing fever, and a total of 1,909 cases of continued and remittent fever in prisoners, 247 of whom died, it is evident that Dr. Cunningham did not support the typhus doctrine.

PESTILENTIA—PALI PLAGUE—MAHAMURREE.

That eminent authority on Indian diseases, Dr. Morehead, strongly objected to these indefinite and separate designations of what appears to be one and the same disease—*i.e.* INDIAN PLAGUE.

Seeing that the existence of this dire malady, either in Western India or on the spurs of the Himalayas, can be traced, with occasional apparent breaks of ten or fifteen years, from 1815 to 1876, and that a full and continuous history of its progress during these fifty-one years appears to be wanting, I shall deal with this subject rather fully. I need hardly say that we can only follow the progress of Indian plague with a good map of India before us. The earliest accounts of this disease are given by Mr. McAdam.² Its first appearance of which we have any full record was in the Provinces of Kutch and Kattywar in the Bombay Presidency in May 1815.

It appears to have been preceded by three years of severe famine. It spread extensively and committed great ravages. Many thought

¹ The italics are mine.—N. C.

² *Transactions of the Bombay Medical and Physical Society*, vol. i.; and an admirable review of the history of Mahamurree up to the year 1852, in the *I. A.*, No. 2, p. 609, and No. 3, p. 199.

that it had been imported from Egypt, but facts are wanting in support of this opinion, and it has been justly remarked that, had it been so introduced, it is probable that its first appearance would have been at Bombay, in the direct line of the greatest traffic.¹ It prevailed chiefly among the dealers in raw cotton (which probably acted as a focus of contagion) and the manufacturers of cotton cloths. Mr. Whyte describes² the habits of these people as being mainly instrumental in causing the existence of this plague. They were the only people he had seen in India who might properly be termed filthy. Ablution, so general in India, was almost wholly neglected in Kattywar. The people were loaded with clothes, which were seldom changed, and which almost rotted upon the persons of the poor. Their immense herds of cattle were invariably driven within the town walls at sunset, and penned at night in the house which contained the rest of the family. Mr. Whyte made a remark which, in our present knowledge of these epidemics, is full of important significance: 'Many of these cattle are labouring under diseases regarding the nature of which we are ignorant. An epidemic or contagious disease has, *since the famine*,³ run through the whole of them.' He considered that marsh poison was not operative here, the towns being situated either on the rocky bank of a running stream or on dry sandy soil. He apprehended that it would be difficult to find a situation more likely to give rise to malignant typhus, as represented by 'jail,' 'hospital,' or 'ship' fever, than many of these walled and crowded towns furnished. The habit of this disease appears to have been to begin in the cold and to last through the hot months, without undergoing any change from the change of weather. It was undoubtedly contagious, and, making full allowance for exaggeration in popular reports, it was very prevalent and exceedingly deadly. The symptoms were those of a low typhus fever with buboes, little inclined to suppurate, in the groin or armpit, less frequently in the parotid and submaxillary glands on one side. In all there was the greatest debility; coma generally set in on the second day. The tongue was covered with a white fur in the centre, through which and at the edges were seen papillæ of a shining, fiery red appearance. Petechiæ were not observed, and I do not find mention of hæmoptysis or any other hæmorrhage.

It is shown, in the article which I am quoting, that the Indian plague prevailed in Kutch, Kattywar, and Ahmedabad from 1815 to 1819 [elsewhere its historian says that it was last known to exist in the Bombay Presidency in 1821]; after which we hear no more of it, in that part of India, until it broke out in the town of Pali, in Marwar, in 1836; but, as I shall presently have to show, its first known out-

¹ Reviewer, *I. A.*

² *Transactions of the Bombay Medical and Physical Society*, vol. i.

³ The italics are mine.—N. C.

break, as mahamurree, in the district of Gurhwal, in the Province of Kumaon, has been traced to the year 1823, from which date we are able to follow its appearances, at intervals, in those hill districts at least up to 1876.

The Pali Plague of 1836.—It was stated that, although this was popularly considered as a new disease, it had formerly committed great ravages in Marwar. For the history of the Pali outbreak of Indian plague we are chiefly indebted to the published report of Dr. Ranken, then officiating secretary to the Medical Board of Bengal.

This malignant fever made its appearance in the town of Pali in July 1836. At a meeting of the Epidemiological Society, in June 1880, Dr. Don gave a vivid description of the insanitary condition in which Pali was when he was stationed in that neighbourhood twenty years previously. He spoke of it as the filthiest native town that he had ever seen, densely crowded within the circuit of a ditch of black and filthy water. Even the natives looked upon it as a peculiarly unhealthy spot, in which pestilential disease might be expected to arise at any time. He thought that, being in a native State, its condition is doubtless still as bad as ever.

After spreading to twenty-four places within a circuit of thirty miles around Pali, the disease reached the city of Joudpore in October. We are told that, having thus begun in Marwar, the disease, affecting only the village of Dewair in the intervening hilly territory of Mhairwarah, passed over it, in March 1837, to Deoghur in Meywar. Thence it skirted the Mhairwarah Hills for fifty miles to Jalia and Ramghur in the district of Ajmere; and, during April, extended in another direction to Bilwarah and Humeerghur on the road between Neemuch and Nusseerabad. Thirty-two villages of Meywar were finally attacked by the epidemic. It did not advance beyond the boundaries of Marwar and Meywar, except at Jalia and Ramghur. It began to decline with the commencement of 1837, and almost disappeared towards the end of that year, without progressing beyond the districts originally assailed. In November of that year, however, according to Assistant-Surgeon Forbes,¹ it again became severe at Pali, and continued up to February 1838.

Medical officers ascertained that it was infectious, and considered that it was the plague.

It is stated that, of the fifteen or twenty thousand inhabitants of Pali, four thousand perished, at the rate of fifty or sixty daily. The deaths in Marwar were reckoned at twenty thousand, including six thousand in the city of Joudpore. In Meywar, the number of victims was computed at seven thousand and twenty-two. The medical officers concurred in stating that not more than one-third of the sufferers survived.

¹ *Transactions of the Bombay Medical and Physical Society*, vol. ii. p. 1.

Mr. MacLean and Dr. Irvine gave the following symptoms as characteristic of the Pali disease. No sense of indisposition gave warning of its approach. It came on with slight rigor, headache, nausea, and pain in the loins. The skin soon became hot and dry, the pulse from 130 to 150, soft and easily compressible. Tongue variously covered with white, light brown, or darkish fur, these colours being sometimes intermixed; no red points appeared. Vomiting and irritability of stomach occurred but rarely. Bowels bound; abdomen tumid and full, but seldom painful on pressure. Eyes heavy, hazy, and bloodshot, sometimes appearing as if injected with lake. The countenance expressive of anxiety and inward pain. Glandular swellings in the groins, armpits, and neck, most frequently on the left side, as also under the jaws and ears and in the upper part of the thigh. These were generally perceptible on the first or second day, and rarely became larger than a walnut; but, in some instances, they became much larger, burst, and discharged pus. The symptoms were sometimes all so mild that the sick kept walking about until they gradually recovered. In most cases, there was a visible abatement of the disease every twenty-four hours towards morning. But, in the worst forms, intense fever continued night and day without any remission: the patients could not rise from their cots on account of extreme debility, and an attempt to raise them to the erect posture produced fainting. Although apparently unaffected by the fever, respiration was often impeded by concomitant inflammation of the lungs. Hæmorrhage from the lungs took place in a few cases, and was much dreaded. Upon the presence of lung complication with hæmoptysis in Pali plague and mahamurree (conditions which seldom occurred in true bubo plague), Hirsch and Liebermeister appear mainly to ground their opinion that Indian plague is identical with the Black Death of the fourteenth century). Mr. MacLean wrote: 'In a small proportion of cases, inflammation of the lungs comes on, on the first or second day of the disease. The patient complains of acute distress on one or other side, or behind the sternum, great difficulty in breathing, and short dry cough. Usually on the second or third day, a small quantity (rarely more than half an ounce) of florid blood in small coagula is expectorated. In such cases, buboes are not commonly observed; though they do occasionally coexist with the inflammation of the lungs. The mortality has been so great among those in whom the lungs were affected, that a person now, on seeing blood in his sputum, gives himself up for lost. There was most excruciating headache. Delirium rarely occurred in the beginning, but coma generally supervened shortly before death. When violent, the malady had its fatal course in three days. When mild, with or without the affection of the glands, it was protracted to fifteen or twenty days.

Some attributed this outbreak to imported contagion; but this

opinion seems to have been unsupported by any conclusive evidence. Mr. MacLean, the first medical man who saw the disease, appears to have considered that the pestilence originated in the filth and bad ventilation of Pali itself.

*Mahamurree, 1823-1876.*¹—Dr. Francis shows that mahamurree was first brought to the notice of Government in 1836, the very year in which Pali plague first attracted public attention. Its first recorded outbreak, however, was in the district of Gurhwal, in the Province of Kumaon, in the year 1823. It subsequently prevailed endemically in some part or other of Gurhwal, occasionally with great violence. Dr. Renny found that it began near Kedarnath in the snowy range. The highest point at which it raged appears to have been the village of Sarkote, situated at an altitude of more than 7,000 feet above the sea-level. Dr. Francis shows that, in 1834, an outbreak of this pest caused 633 deaths in Gurhwal—a large mortality in so thinly populated a district. In 1849-50, 111 deaths took place in nine villages. The maximum death-rate was estimated at 88, and the minimum at 25, per cent. In 1852 the worst outbreak occurred. The disease then burst forth in twenty villages, which were almost simultaneously attacked. Drs. Francis and Pearson were appointed to investigate the malady in 1852. The chief symptoms, as given by Dr. Francis, were as follow. The attack usually commenced with shivering. There was rapid and extreme prostration, with præcordial oppression, glandular swellings, either inguinal, femoral, axillary, or cervical, or immediately below the ear, the suppuration of which was regarded as critical. A white chalky, laterally fissured, but unswollen tongue, occasionally resembling the strawberry tongue of scarlatina. Head-ache, not infrequently accompanied by passive delirium and imaginary noises within the brain; a peculiar mixture of muddiness and lustre in the eyes. There was generally constipation; now and then diarrhœa. The supervention of profuse perspiration was considered highly favourable. Vomiting was not frequent, but the natives thought well of it when it occurred. It was not at all uncommon for an individual to be suffering in the morning from nothing apparently but malaise; yet, in twenty-four hours, the sufferer would be a corpse. In the greater number of fatal cases, death occurred on the third day. Petechiæ were rarely observed. Dr. Francis's allusion to the important question of *hæmorrhages*, which we have spoken of above as occurring in Pali plague, is not very full. He considers that they

¹ The principal authorities on the subject of Mahamurree are: *Medical Report on the Mahamurree in Gurhwal, in 1849-50*, by Dr. C. Renny; the article on Mahamurree in No. 2 of the *I. A.* already cited (this gives the very able report on this disease by Drs. Francis and Pearson), and the former officer's memoir on 'Endemic Plague in India,' *Transactions of the Epidemiological Society of London*, vol. iv. part 3, p. 391.

were not very common. 'These,' he writes, 'are met with, if at all, in the quickly fatal cases; but hæmorrhage is not an ordinary accompaniment. When seen, it is in the form of hæmoptysis.' In noting the fact that the rats of the village almost invariably die on the eve of an outbreak of mahamurree—a circumstance which also occurs in Yunnan, in Western China, where plague has been endemic for some years past—Dr. Francis says that the animal 'would emerge from its hole on to the floor, stagger, perform an involuntary gyration or two, bring up blood, and die.' In the lungs of a rat which the reporter examined, 'there were on the surface what looked like carbonaceous patches—*islands of hæmorrhagic effusion.*'

Dr. Francis and the great majority of observers who have investigated mahamurree in its habitat, place the causation of this plague in the filthy mode of life of the people, the paucity of their vegetable diet, and the absolutely insanitary state of their villages. The picture which Dr. Francis draws of the mode of life of these natives is very striking. He writes (p. 400): 'Until hygienic measures were adopted, the general uncleanness of the people in their persons and *entourage* was incredible. A small stone dwelling (built upon a surface thirteen feet square), consisting of two rooms, each about five feet high, one above another—the upper chimneyless and practically windowless; tenanted by the entire family, often more than half a dozen in number, and by the huge baskets containing the family grain; the lower compartment (a wooden floor, full of cracks serving as media for the effluvium from below, dividing the two) being occupied by the family herds, consisting of cows, goats, and pigs; a row of such dwellings (sometimes they are single or double, spread over an irregular surface) similarly tenanted, and flanked at either extremity by the ancestral heap of manure, from which streamlets of liquid filth were flowing in different directions; the cottages covered with cucurbitaceous creepers; a small forest of hemp, some eight or ten feet high, luxuriating in the immediate neighbourhood of the village; a growth of underwood, including nettles, &c., between the two, and more or less surrounding the latter; and unwashed paterfamilias, seated in front of his fig-tree, having submitted his head to be divested by a faithful spouse of the light infantry skirmishing in his unkempt hair! Conceive such a village, situated towards the base of a mountainous slope, well within the range of whatever noxious influences may emanate from the valley below; located where there would be the veriest minimum of ventilation; and we cannot be surprised if, when sickness does come, it should run rampant. The atmosphere and peculiar smell in these locations must be *encountered* to be appreciated. They are *sui generis*, and are very suggestive of disease.'

Dr. Francis concludes that mahamurree is highly contagious (he uses the term synonymously with infectious), and that, once in exist-

ence, it is rapidly propagated from person to person and from place to place, within certain ascertained bounds.

From the time at which the late Dr. Pearson was first deputed to investigate this disease with Dr. Francis in 1852, he, as holding an appointment in the district, is said by the latter authority to have had experience of mahamurree, which has extended over more than a quarter of a century. In the Thirteenth Annual Report for 1876 of the Sanitary Commissioner with the Government of India, p. 120, I find the following brief observation: 'The recurrence of mahamari in the hill districts of Kumaon and Gurhwal may be noticed. Dr. Planck, the Sanitary Commissioner for the N.W.P., made a thorough inspection of the villages in which this disease occurred, and has given in his annual report a full and interesting account of the outbreak and of the nature of the disease. A short note on the same subject is also furnished by Dr. Watson, Superintendent of Vaccination' (Dr. Pearson's successor). 'Both officers agree in the opinion that in its nature the disease is identical with Egyptian plague. Epidemic outbreaks have occurred previously at intervals of from ten to fifteen years; but, notwithstanding that it spreads rapidly by infection among the hill people, it has never spread to the population of the plains below. Dr. Planck is disposed to attribute the development of the disease to the insanitary condition of the villages, especially to the custom which prevails among the people of keeping their cattle in the same houses in which they themselves live. Dr. Watson, on the other hand, is of opinion that the disease arises from the use of decaying grain, especially mundua (*Eleusine coracana*), a grain much used by the people of the district, in which a peculiar fungus is generated.' Upon this slight statement of its premises it is impossible to argue this opinion that plague is caused by eating diseased grain. The existence of this idea was known to Drs. Francis and Pearson, who consequently made examination of suspected grain and found that it was good throughout. Dr. Francis very much doubts that it ever lights up mahamurree.¹ The entire history of these maladies shows that no European has ever fallen a victim to Indian plague.

Although several commentators have argued with considerable earnestness that this disease, in its several outbreaks since the year 1815, was not identical with the true Levantine plague (as it doubtless was not, no two fevers of like denomination being *perfectly identical in type* when they occur in localities having different climates and situated far apart), the general consensus of opinion has always been that these Indian fevers are true bubonic plague.

In the reports and monographs which I have cited, many pages have been devoted to the question—Was plague conveyed from Egypt

¹ 'Endemic Plague in India,' *Transactions of the Epidemiological Society of London*, vol. iv. part 3, p. 402.

or elsewhere to India, and was it again conveyed from Western India to Gurhwal? The evidence, which is mainly slight and inconclusive, lies there fairly open to anyone who may desire to sift it. I have done so with care and considerable interest; but I do not think that the generality of my readers would be willing to do so in the absence of any hope of arriving at certainty. All who have had the best opportunities of judging have concurred in the belief that, wherever this plague has appeared, the *local causes* were such as to account for its outbreak; and that, although it may possibly have been brought by contagion from a distance, there is no proof that such was the case, and that probability favours a negative opinion.

I do not find that any measures were adopted by the authorities with a view to limiting the spread of the first outbreak of this plague in Western India in 1815-21, but we are told that, when it made its appearance among the Boras (manufacturers of cotton cloth) of Wunkaneer, it prevailed only in that class. Everyone who was attacked is said to have died. The Boras all left the town and went to live upon the mountain in the neighbourhood, leaving only those who were sick in their houses, and one attendant upon each of them. The whole of these soon died; but the pestilence lasted no longer than fifteen days.

It was considered at the time that energetic quarantine arrangements kept the Pali plague almost entirely out of Mhairwarah. This is a hilly district, ninety miles in length, lying between Marwar and Meywar, in which two districts the plague attacked every village, Mhairwarah appearing to be exposed on both sides, throughout its whole extent, to the invasion of the disease. Captain Dixon, superintendent, began by drawing two watchmen from each village and posting them in a continuous chain, first towards Marwar, and finally, when the disease passed into Meywar, along the whole 'double frontier.' These guards of the public health were kept on the alert by peshkars, acting as superintendents, who went their rounds once in two days; they were reported to have successfully opposed entrance by the known routes. But some traders, it is said, persuading the Marwarees to conduct porters with wares for sale through by-ways in the hills to Dewair, infected that village. No other spot in this district suffered. Other sanitary cordons, superintended by medical officers, and patrolled by a thousand horsemen, were established in the north and south of Meywar. The principal military stations of our troops in the vicinity of the affected districts, Ajmere, Nusseerabad, Neemuch, were carefully defended by preventive lines. The able reviewer whom I have so largely quoted adds: 'Whether from these energetic measures of precaution, or from some inscrutable law of the disease, it almost disappeared towards the end of the year 1837, without progressing beyond the districts originally assailed.'

Although this has every appearance of having been an indisputable

example of triumphant success in the experiment of checking the progress of an extremely communicable pest by means of sanitary cordons, and although it cannot be denied that this measure may very probably have been useful, we must perceive that other causes—locality, temperature, climate, soil, the diet and habits of the inhabitants, and their general hygienic surroundings—may probably have been very far more efficient in protecting the exempted localities. Dr. Pearson informed Dr. Francis that he has never heard of the exportation of mahamurree since they worked together in 1852–53, save that, many years ago, a disease having very much the general character of mahamurree appeared in one or two insanitary villages at the immediate foot of the hills in which mahamurree was raging. Every Indian physician has probably followed with deep interest Dr. Edward Balfour's inquiry for localities in Madras exempt from cholera. Dr. Francis shows that the progress of mahamurree is marked by what appears to be a like 'caprice,' but which is probably governed by fixed natural laws still open to scientific inquiry. Thus Dr. Francis tells us of two villages, both situated on the same mountain steep and embedded in the same jungle, with the same N.N.E. aspect, only five hundred yards apart; and yet, at every visitation of mahamurree, whilst one has always escaped, the other, Kunowlee, has not. 'Doubtless,' Dr. Francis adds, 'there is some condition, the nature of which has not been fathomed, that secures the exemption.' I wrote, at the time—'the mystery would be resolved should my friend Dr. Planck's local correspondent report that these two hamlets are respectively supplied by rival milk or water companies.'

Systems of quarantine and the employment of expensive and cumbrous sanitary cordons have never been demanded in the management of the plague of Gurhwal. This pest can scarcely be said to have *spread* at all. It self-evidently depends upon such exclusively local causes that it might, strictly speaking, be styled not so much 'endemic' as 'sporadic.' The atmospheric or telluric endemia spares clean villages, but smites dirty ones. Attention to the commonest rules of personal, household, and village cleanliness has invariably put down every outbreak. The enforcement of these, however, from the first, always demanded scientific European supervision, carried out with absolute strictness and energy. The natives are inactive, incredulous, and disobedient. Thus Dr. Francis tells us that, at the outset, it was only when Dr. Pearson was invested with magisterial authority and power to enforce his own orders, up to the point of burning the infected villages, that his directions were complied with.¹

¹ For a fuller account of the sanitary measures adopted, see my work on 'The Means of Preserving the Health of European Soldiers in India,' *I. A.*, No. 12, for 1860, p. 614. They were effected at an expense to the State of only Rupees 1934.11.6—less than two hundred pounds sterling.

As might be anticipated, little can be said with regard to the *Treatment* of cases of Indian plague. Quinine has not the power of arresting the disease ; but here, doubtless, as in true enteric fever and in nearly all Indian diseases, its moderate but steady use, as a means of sustaining vitality, is clearly indicated. Dr. Francis agrees with Dr. Watson that hyposulphites 'would, doubtless, be found useful.' Good hygiene and supporting diet are equally indicated ; but, of course, in those localities, the difficulty has always been to find cases of this rapidly deadly fever so little advanced as to afford fair scope for medicinal treatment.

In speaking of Indian relapsing fever, I shall allude to the fact that there is a marked coincidence, if not a positive connection, clearly observable in the times of the occurrence of great outbreaks of Indian plague and of Indian relapsing fever. Whether there is or is not an intimate relation between the two diseases—which are, however, absolutely different in type—or whether they are to be regarded as entirely distinct, but concurrent, products of great atmospheric or telluric disturbance, evolving various epidemics, there can be no question that one has always appeared as the almost immediate herald of the other ; and that the two pests have run their own courses of destruction concurrently, but usually in different and often widely separated districts of India. To follow this question with precision, I must beg the reader to refer to my next chapter on Relapsing Fever.¹

There have been, within our knowledge, four great or singularly marked outbreaks of Indian plague :

1815-21	.	.	.	In Guzerat, Kutch, and Kattywar. In 1823, in Gurhwal.
1836	.	.	.	In Pali, Meywar, Malwa, &c. ; also in Gurhwal.
1852	.	.	.	In Gurhwal.
1876	.	.	.	In Gurhwal.

In 1816 . . . while Indian plague was prevalent in Western India, a most destructive outbreak of relapsing fever raged between Patna and Seharunpore in the north-west. I have cited Dr. Jamieson's account at p. 97. In 1817, the great epidemic outbreak of cholera ravaged Bengal. Thus, during the first three years of the prevalence of plague in Western India, the entire breadth of British India, from west to east, was swept by pestilence. In 1818, while plague was still prevalent in Western India, relapsing fever was distinctly observed at Bareilly—page 98.

In 1836 . . . when plague broke out at Pali, mahamurree 'first attracted much notice' in Gurhwal (Renny) ; and Bareilly, Rohilkund, Mynpoori, and the districts around Delhi were ravaged by relapsing fever—p. 98.

In 1852-53 . . . Drs. Francis and Pearson were directed by

¹ *M. T.*, p. 115 and 145 of vol. i. for 1880.

Government to investigate that which Dr. Francis designates as 'the worst outbreak' of mahamurree in Gurhwal. In these years occurred the epidemic of relapsing fever in the Usufzaie Valley about forty miles from Peshawur, described after Dr. Lyall at p. 82 and 96.¹

In 1876 there was an outbreak of mahamurree at Gurhwal, described by Drs. Planck and Watson, as cited above. In April 1877 commenced the outbreak of relapsing fever, which was the first manifestation of this disease observed in Bombay, and of which an account is given by Dr. H. Vandyke Carter in his recently published monograph on spirillum fever.

It was observed by Dr. Robert H. Irvine² that, during the height of the Pali plague, at the places infected, cattle, sheep, and dogs suffered and died in numbers, from fever and enlarged glands in the throat and neck. In some animals which he observed at the time, the swelling of the sub-maxillary glands was the only affection. He added that similar diseases of animals have been often observed in the Levant and other places during plagues. In the same way, during severe cholera, domestic fowls often die very suddenly in great numbers and apparently of that disease. At Hoshungabad, he frequently observed fowls, during severe cholera, turn giddy, pass watery stools and speedily die.

INDIAN RELAPSING FEVER.³

The question—Is Relapsing Fever a new disease in India?—may, perhaps, be still open to controversy, but I think that we have strong evidence upon which to base the conclusion that it is one of the indigenous pests of the country, and that its history may be traced back at least to the middle of last century.

Relapsing fever having been clearly recognised in Europe, especially by Drs. Henderson and Wardell, as a disease altogether distinct from typhus, in 1842, when it became generally prevalent throughout the United Kingdom, was first distinctly observed and described in India by Dr. Robert Lyall in 1852–53.

¹ It appears probable that the Usufzaie fever formed a link between Indian plague and Indian relapsing fever. It was contagious, very destructive to life, and marked by a tendency to relapse; but it had a feature which is not usually characteristic of Indian relapsing fever, *i.e.* there were inflammation and suppuration of the glands in the groin, axillæ, and neck in some who survived the relapses. I have seen parotid suppuration in typhus at Guy's Hospital, and this frequently occurs in the remittent of the cold season in Calcutta, no other glands appearing to be effected in either case; but, when a malignant, contagious, and deadly fever is attended with inguinal buboes, it becomes allied to true plague. The Usufzaie fever, however, was evidently allied to relapsing fever in being characterised by yellowness of the surface and by relapses.

² *Medical Topography of Ajmeer*, p. 119.

³ Read at the Epidemiological Society, January 7, 1880.

I have given a brief abstract of Dr. Lyall's report of the fever which carried off large numbers of the inhabitants of the Yusufzaie Valley, situated about forty miles from Peshawur, in 1852-53, in the chapter on Typhus, page 82. The following were the chief characteristics of this disease:—It was epidemic, it was distinctly contagious, it was extremely prevalent, out of 226 houses in one village, 131 were found to have been visited by the disease; and, of the 949 inmates of these houses, 411 (or nearly one-half) were attacked by it. This fever presented a characteristic in which the relapsing fever of India has generally been found to differ from that of Europe—it was very destructive to life—out of 410 attacked, 125 had died, and 126 remained sick when the report was made. At the commencement of the epidemic, jaundice was not an invariable complication; as the disease increased in prevalence, the body, in almost every instance, became jaundiced at about the fourth day, and a tendency to diarrhoea showed itself. Dr. Lyall wrote: 'The most remarkable feature in this complaint is its tendency to relapse from two to ten days after the subsidence of the previous attack, without any irregularity on the part of the patient, in the way of exposure or diet, to account for it. These relapses are always attended with great derangement of the bowels, and often seem to depend upon a low degree of gastro-enteritis. A large number of people are carried off from the chronic dysentery arising from this cause. The convalescence in all is remarkably slow, and many months are always necessary before their health becomes anything like re-established.'

There were inflammation and suppuration of the glands in the groin, axillæ, and neck in some who survived the relapses. At the onset of the fever, quinine was not successful, but it was useful in the stage of relapse. The poor, uncleanly, ill-fed, and badly clothed and lodged were the chief sufferers. Thus, in the fever of the Yusufzaie Valley, we have displayed all the most distinctive characteristics of the relapsing fever of India—an extremely prevalent and destructive epidemic or endemic fever, arising among the poor and starved, and perhaps spreading to the well-to-do inhabitants by force of its contagiousness. The tendency to relapse is very marked, the convalescence is remarkably slow, there is generally jaundice, and quinine has no specific power as an antidote to the fever poison.

The fever observed by Dr. Lyall was clearly a disease very nearly allied to the ictero-hepatic form of relapsing fever, the bilious typhoid of Lebert, probably modified, as the continued fevers of India generally are, by the influence of marsh miasm. Thus it was noticed, in this and in other similar outbreaks, that 'the relapses of the fever assumed the remittent or even intermittent type.'¹

¹ Vide Dr. Lyall's and Dr. Farquhar's reports on this fever. *I. A.*, No. 3, p. 16, and No. 4, p. 504. It will be well that readers who are not experienced in the

India was long supposed to be as free from relapsing fever as from true typhus. In the edition of his treatise on continued fevers of 1862, page 301, Murchison said : 'In India, and indeed in all tropical countries, relapsing fever is unknown.' Other leading authorities maintained the same opinion. But, in his edition of 1873, page 328, Murchison fully admitted the occurrence of this disease in Upper India.

We have strong grounds for believing that the disease which Dr. Lyall first recognised in 1852 as relapsing fever was then by no means a malady new to India.

In his report on cholera, Mr. Jamieson has given a vivid narrative of an outbreak which ravaged Upper India in 1816. Throughout the Upper Provinces, there prevailed most extraordinary drought and heat—such as had not been experienced since 1803. To the long-continued drought succeeded heavy and incessant rain, so that the whole face of the country was laid under water. The disease appeared among the natives in August, and raged epidemically in every city and town between Patna and Seharunpore. 'It exhibited itself in the shape of a bilious remittent fever of a violent inflammatory type, accompanied, like the yellow fever of the West Indies, with suffusion of the skin.' Its tendency was to terminate fatally at the end of two or three days. 'The width of its range precluded the possibility of its being referred to any causes purely local.' Its attacks were indiscriminate ; 'it seized equally Europeans and natives, and as readily entered the open and spacious house of the officer and civil servant as the crowded barrack of the soldier' [barrack accommodation in India was then generally very faulty] 'and the low filthy hovel of the native.' The mortality in the largest cities was very great. 'Of numerous native villages nearly the whole population was ill at one and the same moment, and many of the shops were shut for want of people to attend them. The banks of the river were covered with the dead and the dying.' In Delhi, of two native corps alone, there were five hundred men at once sick in hospital.' The European troops suffered quite as severely. 'Of the European flank battalion, 648 strong, stationed at Allahabad, 305 men were attacked during October. Early in November, there were at one time 180 rank and file and twenty women and children on the sick list ; and, in the whole, only seventy men of the seven companies of which the corps consisted escaped the disease. But Cawnpore was the principal seat of its ravages amongst Europeans : of four King's corps, the 24th Regiment L.D., 735 strong, and the 14th, 66th, 87th Regiments of Foot, 852, 813, and 702 strong, then in cantonments, there were, from first to last, nearly one thousand persons taken ill. The disease began here in August, prevailed during the three succeed-

diseases of India should, as far as possible, consult and study the originals of the many valuable reports to which I shall have to refer, and of which the scope of my commentary permits me only to give brief abstracts.

ing months, and did not finally withdraw until the setting in of the cold weather in December. It was at its height in September and October; eight or ten, or sometimes even fifteen men then died daily. The 87th and 66th Regiments suffered dreadfully; from the time of their reaching the station, they lost nearly four hundred men. The former corps is said to have had 119 in hospital at once, to have buried twenty-one persons (including women and children) in one day, and upwards of ninety of its number within a month.' 'This,' adds Dr. Jamieson, 'is a degree of mortality far exceeding anything then on record in the medical annals of Bengal.'

Considering that this report was written twenty-six years before relapsing fever was recognised in Europe, and remembering that other Indian fevers are not characterised by a tendency to relapse, the following statement made by Mr. Jamieson must, in connection with the rest of its history, be taken as clearly showing that this malady was Indian relapsing fever. 'It has been stated that the disease abated in December' [it commenced in August]; 'so early as the end of November its attacks had become less frequent, and its symptoms milder; *but relapses were still common*,¹ and it was very difficult to counteract the extreme debility invariably consequent on the attack.'

It is particularly noteworthy that the first visitation of Indian plague prevailed in Western India from 1815 to 1819, and that the outbreak of relapsing fever described by Jamieson occurred in the Upper Provinces only a few months previous to the commencement in Bengal of the great cholera epidemic of 1817. So also when, in 1836, the Pali plague, commencing in Marwar, spread rapidly and attacked thirty-two villages of Meywar, a destructive fever, which must have been relapsing fever, made its appearance in the town of Bareilly,² and committed several ravages in Rohilkund, in Mynpoori, and in the districts around Delhi. In tracing the history of relapsing fever in India, it is interesting to observe that, in his narrative of the outbreak of this disease at Bareilly, Mr. Guthrie states that: 'It is reported that about the year 1818 [when, it must be repeated, Pali plague prevailed in Western India], during the medical charge of Mr. Evans, a similar fever prevailed, sacrificing 300 victims; reappearing under Mr. Browne in 1818, and causing great mortality from July to September; and that it also paid a passing visit, under Mr. Rhodes, in 1834, of only a few days, destroying eighteen convicts.' The reports show clearly that this disease was contagious, but not so extremely contagious as

¹ The italics are mine.—N. C.

² The history of this disease will be found in the following reports:—Hugh Guthrie (Bareilly, *C. M. T.*, vol. viii. part 2, p. 189; and of Dr. D. Macnab (Mynpoori), *Ibid.* p. 230; and those of Stuart, Spencer (Moradabad), and Shirreff (Delhi territory), in Dr. James Ranken's 'Report on the Malignant Fever called the Pali Plague,' pp. 193 *et seq.*

the fever of 1816. Mr. Guthrie says on this point : ' As to its infectious character here, within certain limits, I have no doubt but it has laws of its own. When the town was devastated by it, the convicts had it not among them, but now it is *vice versa*.' Jaundice usually characterised this fever, the eyes being of a deep saffron colour. The disease was not, at its outset, amenable to quinine ; severe pains in the bones were grievously complained of ; there was great tendency to bowel irritation ; recovery was extremely slow. The malady prevailed almost exclusively among the poor and among prisoners crowded into ill-constructed and deficiently provided jails. Dr. Stewart, of Moradabad, wrote : ' Persons living in well-ventilated places entirely escaped this epidemic,' and ' Persons who could afford themselves good food, even though living in indifferently ventilated situations, provided they avoided exposure to the infection, also escaped.' Most of the medical officers reported very unreservedly upon ' the ill-housed, overcrowded, badly clothed, and ill-fed condition of the prisoners.' The destruction of life was very great. Mr. Shirreff reported that, at one village in the Panipat district, nearly two hundred persons died, out of a population of five hundred families, within six weeks ; at another place, two hundred and thirty were said to have died within twenty-seven days out of a population of some one thousand inhabitants. In one division of this village, almost every one who was attacked died. Between October and February, inclusive, the monthly range of deaths was from nineteen to thirty-two, out of a body of sick averaging from ninety to two hundred and forty-five. Considering that, at that time, relapsing fever had not been differentiated, the evidence that this was the relapsing fever of India is very strong. Shirreff noticed that, in the Panipat and Rhotuck districts : ' In the majority of cases the fever runs its course till the seventh, eighth, or ninth day.' Macnab says that, at Mynpoori : ' When the disorder proved fatal, the issue did not long remain doubtful ; the course towards this consummation was run over rapidly, and five or six days may be given as the maximum period.' Some were dealt with much more summarily. Spencer says, in describing the disease at Moradabad : ' The patient died sometimes on the third, but more commonly on the fourth to the sixth day.' It is to be borne in mind that, unlike the allied disease in Europe, the relapsing fever of India is of such extreme deadliness that it frequently destroys the patient so early that the tendency to relapse cannot be observed in many cases. Thus the reports show that many died early in the disease from head, chest, and abdominal complications ; and Guthrie wrote : ' Many die in the first paroxysm, completely overpowered, as if the powers both of body and mind were at once blighted. This tendency to sudden death, from shock, immediately the malady, in its overwhelming power, strikes the patient down, has been observed in plague, in cholera, and in variola.' Spencer, describing the Morad-

abad fever, makes a statement which, standing alone, might be taken as sufficient evidence that this malady was relapsing fever; he says, 'Many men suffered severe relapses which mostly terminated fatally;' but, while I consider that our mass of evidence in favour of this view is quite weighty enough to enable us to dispense with this striking statement, it is, I think, clear from the context that the writer used the expression 'relapses' as synonymous with 'sequelæ,' which were here dysentery and large abscesses. However, the evidence of Guthrie and Macnab on this point is very strong and direct. Mr. Guthrie had a convalescent hospital where those discharged from his fever hospital were accommodated and placed under the care of an assistant, who was ordered '*instantly, as relapses occurred, to send them back to the hospital bungalow*'; from these again, as they recovered a little strength and power of digestion, some were selected daily, and placed in another outhouse under the care of the native doctor of the Insane Hospital, who saw them fed, exercised, &c., and, if these *did not in five or six days suffer a relapse*, they were sent to the jail as cured. The grand difficulty was to restrain them from eating, as the indulgence in even a moderate quantity of solid food *re-induced the fever or caused fluxes*.'¹

Macnab wrote: 'Low delirium was very common, and when a torpor supervened, the case might be considered hopeless. We at times, however, had cases where the stupor partially vanished, and the intellect became in some degree clear; but this gleam was evanescent, for suddenly a relapse would dissipate the illusive hope, and the patient fall back into a state worse than that from which he had partially emerged. If, during the first day or two of improvement, the slightest retrograde movement were made, hardly an individual recovered.'

The above facts convey a very important lesson to the young practitioner. He must not resolve to deny that a fever which suddenly appears among the poor of the town, or the prisoners in his jail, is Indian relapsing fever—solely upon the ground that none of his earlier cases are marked by anything like the cessation of fever followed by a distinct relapse of fever. If the type is nearly that of an extremely severe remittent, with or without jaundice, and not amenable to quinine, and if there be substantial evidence of contagion, and the spirillum be detected in the blood, the disease is relapsing fever. Still it is clear that, whenever an outbreak of Indian relapsing fever is carefully observed throughout its course, positive evidence of the existence of the characteristic tendency to relapse will not fail to present itself.

As I have already mentioned, Dr. Joseph Ewart's experience when attached to the Meywar Bheel Corps, after he had observed cases of

¹ The italics are mine. —N. C.

typhus and true enteric fever in the Ajmeer jail in 1855, led him to inquire, in the 'Indian Lancet,' whether many of the so-called malarious remittents are not, in reality, typhus, typhoid, and relapsing fever.

The last commentary on Indian fevers published by Dr. Kenneth Mackinnon was a note on 'Fevers with Yellowness of Skin,' published in the 'Indian Annals of Medical Science,' No. 7, 1857, page 155. It may be taken as a useful lesson to many of us to observe how—in reviewing, for a second time, Jamieson's narrative of the epidemic fever of 1816, and the accounts of the pestilential fever of 1836–37, and comparing this disease with the Yusufzaie fever; and while manifestly yielding to the force of evidence towards the conclusion that these were outbreaks of relapsing fever, and frankly admitting that they could, at most, have been, if of miasmatic origin at all, only in part due to paludal influence—this admirable observer and absolutely truthful reasoner still clings to his old traditions, arguing that marsh poison acted here in combination with animal and vegetable filth; that Dr. Maxwell appeared not to consider the fever he saw at Dera Ghazee Khan contagious; that yellowness of the skin, glandular swellings, and hæmorrhage, which attended these fevers, assimilate them more to the plague than to any other disease; and adroitly urging that this deadly Indian fever can scarcely be identical with the by no means destructive relapsing fever of Europe described by Jenner. A careful perusal of these four most interesting pages will, however, show that his opinions had begun to change; and that, in all probability, he would have been among the first to recognise the true nature of the relapsing fever which made its appearance in the jails of the North-Western Provinces in 1860.

Several years, however, had still to elapse before the occurrence of relapsing fever in the jails of Upper India was generally suspected. A very destructive contagious fever appeared in the Central Jail at Agra in 1860, and was described by Dr. Walker. It again prevailed, in a very deadly form, in the Agra jail in 1864; and, in that and the following year, it appeared in the jails of Umballa, Futtehghur, Allyghur, Jyepore, Malwa, &c. It was described by the several reporters as true typhus, contagious fever, typhoid fever, and, in the words of the Sanitary Report: 'Some, again, believe it to be identical with the relapsing fever of Europe.' Dr. Gray, Dr. Bateson, and Dr. Dallas clearly recognised this disease, as it occurred at Agra, Lahore, and Umballa, as relapsing fever. The whole question was most fully and ably reviewed by Dr. David Boyes Smith in the 'I. M. G.' for 1867 and 1868; and, although his verdict met with a good deal of opposition (especially from the medical side of the Indian Sanitary Commission), he, I consider, brought forward very strong arguments in favour of the opinion that the contagious fever of the up-country jails was nearly identical with the relapsing fever of Ireland, Scotland, Germany,

and America. Indeed the Sanitary Commission went so far as to admit that 'the disease resembles the famine fever of Europe in many important particulars.'

As we have already seen, the relapsing fever of India is distinguished from the European disease in being attended with great mortality. The fever at Agra killed 406 prisoners in 1860, and 250 in 1864. The opponents of the view held by Lyall, Ewart, Gray, Bateson, and Smith insist strongly upon this point, and also deny that famine had anything to do with the causation of this disease in the Indian jails. These arguments are met by the facts that, although it is not a matter of necessity that an Indian famine should be attended with relapsing fever—the last famine of 1876–77 in the Madras Presidency had not this aggravation *in that Presidency*, although it had in Bombay—the relapsing fever of India always originates either among the poor or the prisoners, and that, at least in the Punjab, the Lieutenant-Governor, Dr. Dallas, the Inspector-General of Jails, and Dr. Gray so fully concurred in the belief that the appearance of this fever was clear evidence of a bad hygienic condition of the jails, that orders were given making great improvement in the diet, lodging, and clothing of the prisoners.

It can hardly be expected that men who examine this fever from a scientific point of view, by the light of Indian experience, will be shaken by the personal and political arguments that this is not relapsing fever because it devastates certain districts in which famine has not recently occurred, or destroys 25 per cent. of the prisoners in several central jails under the eye of a humane government and of officers whose administration has been reduced to a system which is nearly perfect. I have already argued (p. 24) that the poor of India, who are the descendants of many generations of starvelings, are, by reason of the deficiency of nutritive elements in their food, subject to the life-long misery of chronic famine. Of these poor the majority of the jail population are the most singularly unhappy representatives. The axiom, generally applied to the European criminal, that he represents the worst blood, the largest sum of constitutional disease, and the lowest feeding, clothing, and lodging discoverable in his city, is equally true of the ordinary Indian prisoner whom destitution frequently precipitates into crime. Again, although it may have long been true that the food allowed in an Indian jail is somewhat better and more ample than that obtainable by the very poorest free inhabitants of the district, its power of maintaining health under the amount of labour exacted is not always unquestionable. There still, again, remains the very serious question—do the prisoners actually obtain the authorised allowance? In reply to this I may give one or two facts which were painfully forced upon my notice during twenty-six months in which I officiated as Inspector-General of the fifty-two

jails in Bengal. I may premise that, at my first large civil station, Chittagong, I once arrested the attendants as they were carrying out of the front-door of the jail, in broad daylight, close past the lodge where the darogah (native jailor) usually sat, a huge basket of hot rice. When I reported this occurrence to the magistrate, he appears to have held some inquiry, the result of which was that it was intimated to me that he had ascertained that the prisoners were accustomed to send what remained from their meals for the support of a favourite Brahmany bull which infested the neighbourhood of the jail. To this I felt constrained to reply that the quantity of rice which I had seen was, self-evidently, in excess of the gastric capacity of any Brahmany bull, however voracious; that the grain was not leavings, but perfectly white rice fresh from the cooking vessels; and, further, that my acquaintance with the physiology of the Brahmany bull taught me that, however capable that sacred animal might be of eating an immoderate allowance of simple rice, it was difficult to see to what dietetic use, he being a strict vegetarian and quite above all suspicion of addiction to stimulants, would apply that large allowance of *mar-salla*—composed of dhall, oil, strong spices, and capsicum—which I saw lying upon the heap of snowy rice. Further, that a very large majority of the prisoners were Mussulmans, who care nothing whatever for the Brahmany bull save when he appears before them in the form of appetising *kiwabs*. Many years afterwards, when I held temporary charge of the prisons, the superintendent of the Alipore Jail reported that a large batch of convicts had arrived from Midnapore in a starving condition. I immediately wrote to Midnapore, which is about a night's post-journey from Calcutta. On its way, my letter was crossed by one from the very able joint magistrate, informing me that his jailor—one of our pattern jailors—had been detected in robbing the prisoners daily of large basketfuls of cooked rice. Soon after this I sent a circular to magistrates, requesting that, at a certain hour on a certain day, they would test all weights used in weighing food in the fifty-two jails of Bengal, by having them weighed against rupees in their presence. The result was that scarcely a single weight came up to the just standard, the weights of the pattern jails being generally most deficient: 'The scales were out of gear,' 'The weights were worn,' &c., &c.! Until you can fill your jails with perfectly healthy, well-nourished felons, and can provide that they shall not be over-tasked and under-fed, you may not, in reason, presume to maintain that, when a contagious fever appears and kills off 25 per cent. of your prisoners, it cannot possibly be of the nature of relapsing or famine fever.

To continue the history of relapsing fever. In April 1877, Mr. Blayney, a medical practitioner and a member of the Town Council of Bombay, stated that a fever which had appeared among those lodged in the relief camp on the flats was not the ordinary malarious (marsh

remittent) fever, but was 'a contagious epidemic disease—a relapsing fever.' The Government immediately desired Surgeon-General Hunter, the head of the medical department, to institute inquiry into this fever. Sir Guyer Hunter appointed Dr. Hewlett, the Acting Sanitary Commissioner and Dr. H. Vandyke Carter, to this duty. The report of the Committee and Sir Guyer Hunter's report appeared in the 'I. M. G.' for October 1, 1877. The Committee's report appears to have been somewhat premature and to have been founded upon insufficient data and observation, but it contained the important announcement that Dr. Carter had discovered in the blood of these fever patients the *spirillum* already recognised as characteristic of the relapsing fever of Europe. Sir Guyer Hunter's opinion was opposed to that of the Committee, he maintaining that evidence was wanted to prove that the disease was relapsing fever. An important report of the whole investigation has been given by Dr. H. Vandyke Carter.¹ Dr. Carter's report may be so readily consulted that it will be sufficient to cite a few of its leading statements. He shows that crowds of indigent peasantry, flocking as refugees from the prevailing famine to Bombay, brought with them not only destitution and over-crowding, but also a contagious fever, which soon spread to the resident population immediately in contact with them, and became the cause of death to many. He regards the disease as famine or relapsing fever. The report is founded upon the examination of about 350 cases of this fever. Quinine had no specific action in this disease. Both at the height and towards the close of the epidemic, Dr. Carter says cases occurred which had all the characters of the so-called 'typhus icterodes' or 'bilious typhus,' in a most pronounced degree. In these cases there was generally 'a yellow tinge in the conjunctivæ.' 'Jaundice was commoner here than it usually is in' (the relapsing fever of) 'Europe.' When at its height, about the seventh or eighth day, the general distress abruptly came to an end with critical sweating and considerable fall of temperature. There occurred from one to four relapses in 50 per cent. of the cases. Abortion in pregnant women occurred in the Bombay fever just as it does in that of Europe. The ravenous appetite, so generally observed in this type of fever, was often noted. The average death-rate was about 10 per cent. of those attacked. A marked characteristic of this fever was the presence of the *spirillum* (*Spirochæte* Ehr.) in the blood, in immense numbers, 'often incalculable except by hundreds of millions,' in some cases, rarely in others. They were often so crowded as to offer mechanical impediment to the circulation, evidenced by dusky lividity of the countenance. The parasite was sought for in many cases of remittent fever and ague, but was not found. It was not found in enteric fever

¹ *Medico-Chirurgical Transactions*, vol. lxi. p. 274, 'Notes on the Spirillum Fever of Bombay,' 1877.

(except in one very doubtful instance) or in small-pox, varicella, or symptomatic fevers. Dr. Carter says: 'Once indeed I found a minute and active organism in the blood of a patient affected with fever, seemingly intermittent in type; but, in general, I conclude that the spirillum must be regarded as distinctive of one clinical pyrexial form.' In the 'Lancet' for June 7, 1879, p. 814, Dr. Carter announced that the spirillum fever may readily be communicated to the *macacus radiatus*, a common quadrumanous inhabitant of the Bombay forests, by inoculation with the blood of a fever patient, or with that of an infected monkey, with the result of marked general symptoms, and abundance of the attendant blood-parasite. Post-mortem appearances, too, are equally accordant.¹

It is well known that Dr. Lyons, one of the most laborious modern investigators of Indian fevers, Mr. Oldham, and others, absolutely deny the existence of paludal poison. Mr. Oldham, however, believes in the separate existence of that Indian remittent fever which most of us characterise as paludal. Dr. Ewart long ago contended that some cases of so-called paludal remittent would, upon closer investigation, be found to be true enteric, typhus, or relapsing fever, complicated with, and masked by, the marsh poison. Dr. Lyons has published two elaborate works² in exposition of his doctrine that Twining, Morehead, and many others of us have been deceived in believing that there is, or ever has been, such an entity as paludal remittent fever in India, but that all such cases, carefully observed, will prove to be examples of continued fever—enteric, relapsing, or typhus. Many of Dr. Lyons's contemporaries will live and die in the conviction that paludal remittent, curable by quinine, is prevalent all over India (otherwise why not do away with quinine?); but we must all feel grateful to Dr. Ewart and to him for the manner in which they have illustrated the necessity of sifting our cases of grave Indian fevers, and of eliminating those of true enteric and of relapsing fever, all of which were, but a few years ago, either entirely confounded with, or, according to the sagacity of the practitioner, more or less imperfectly separated from the 'remittents,' and were invariably reported and embodied as remittents in the returns. A careful perusal of the third section of 'Lyons's Treatise' will lead to the conclusion that relapsing fever has been far more prevalent in the East than was generally believed. Of this we have many illustrative facts before us. To give one example out of a score: Dr. John Macpherson has recently informed me that, during the Burmese War in 1852-53, he received communications from Dr. Murphy, who had served in Africa and in the West Indies, insist-

¹ In 1882 Dr. Carter embodied his observations on this fever in a volume, *Spirillum Fever as seen in Western India*.

² 'Typhoid Fever Discriminated from Remittent Fever,' &c., *I. A.*, No. 28 1871, p. 241), and *A Treatise on Relapsing or Famine Fever*, London, 1872.

ing that a disease which prevailed in the European force in Burmah was perfectly identical with true yellow fever. The reports of the 80th and the 18th Royal Irish Regiments by Messrs. Taylor and Stewart¹ show that, in the first of these regiments, the deaths were 19 to 796 admissions for fever; and that, in the second corps, they were also 19 to 796 admissions for fever. The icteroid character was well marked. Contagion does not appear to have been suspected.

It was, some years ago, well remarked that, whenever a fever case in India puts on an icteritious appearance, especially in a jail, we ought to be on our guard against an outbreak of relapsing fever. Every such case should be thoroughly sifted and closely watched. True jaundice is not, and has not been, as far as I am aware, a characteristic of the paludal remittent of any Indian locality. It is, of course, needful to distinguish the pallid, dusky greenish or yellowish cachæmic aspect presented by many sufferers from the worst paludal fevers from that seen in true icteroid fever. Some years ago I treated several of a party of European workmen, most of whom were attacked with serious remittent fever while setting up an iron bridge at Peealee, on the Mutlah Railway, about thirty miles from Calcutta. The aspect of these patients was so characteristic that I, more than once, on entering the ward and glancing along the beds, said, 'That is a fresh case of Peealee fever!' This, however, was not jaundice; it appeared to be due to disintegrative blood-change.

The question, *Does relapsing fever occur in the Lower Provinces of Bengal and in Calcutta?* is one which still requires careful investigation, even by those of us who have practised nearly all our lives in that part of the country. There is no positive evidence to show that the disease has appeared, within this century, in any town or jail of the Lower Provinces. The result of my own experience of upwards of twenty-seven years in malarious places is that, when I review the subject, my mind reverts to the case of *one* native who was brought into my ward moribund in fever, with saffron-coloured eyes and sordes about the mouth. Kenneth Mackinnon repeatedly insists that he could never trace any evidences of contagiousness in remittent fevers prevalent in and below the Province of Behar. Dr. Francis Anderson's committee, who carefully investigated the Burdwan fever in 1864, were convinced that it was not contagious; and this opinion has, I believe, been held by all who have since observed this disease, with the exception of Dr. Lyons. I, however, think that few Calcutta medical men will be able to concur with him in thinking that Burdwan fever, which he terms 'the non-eruptive disease of Bengal,' 'communicated the eruptive and milder variety' [dengue] 'to the residents of the wealthy and well-cared-for metropolis.'² My friend, Dr.

¹ *I. A.*, No. 2, 1854, pp. 418, 431.

² Treatise, preface, p. 7.

William Palmer, has informed me that he had large opportunities of studying relapsing fever in England, but that, during his long Indian career, a great part of which has been spent in the Calcutta hospitals, he has never seen a case of this disease. Still, the following points appear to claim attention. The fevers with which Calcutta was visited between the time of its first occupation, late in the seventeenth up to the beginning of the present century, were almost unaccountably deadly. We read of 460 burials out of 1,200 British inhabitants of Calcutta between August and the ensuing January,¹ and of a detachment of 240 men, chiefly Europeans, stationed at Fultah, on the Hooghly, of whom not thirty were left alive between August and December.² Dr. James Lind wrote of the fever which prevailed in Bengal in the year 1762, and which he terms 'putrid and remitting marsh fever,' as 'raging more or less in different places according as the soil was more or less swampy,' and as becoming so violent, especially during the rainy season, as to end in death during the third fit, which was generally the case, while others were exposed to the danger of dying at every fit. Admiral Stavorinus, speaking of the fever which prevailed among the European inhabitants of Calcutta between 1766-71, says that 'it generally sweeps away those who are attacked by it in the course of three days.' Dr. John Clark describes 'fever and flux' as being very destructive in Calcutta between 1768 and 1771. He states that fever frequently carried off the patient in twelve hours.³ Assuredly these are not the characters or the death statistics of the paludal remittents of the present day! They more resemble those of the relapsing fever of 1816, in which it was calculated that, between the middle of January and the end of June, there were 8,500 deaths out of a population of about 55,000.⁴ The evil sanitary condition of old Calcutta, the prevalence of sea-scurvy in ships' crews, and inappropriate treatment, prejudicial as they undoubtedly were, are inadequate to account for such death-rates as these. We have very little precise information with regard to the type of these Calcutta fevers; but Dr. Lyons notices that Dr. Lind wrote of the deadly Bengal fever which he observed in 1762, that the 'patients are very subject to relapses;' and also that, in Ives's narrative of the sickness of Admiral Watson, who died in Calcutta on the third day of fever in August 1756 (when the frightful mortality from fever alluded to above was going on in Major Kilpatrick's detachment down the river at Fultah), it is mentioned that 'his eyes were a little yellow.' Further, that, in the 'Selections from the Records of the Calcutta Presidency General Hospital,' published by Mr. Alexander

¹ Captain Hamilton, between the years 1688 and 1723.

² Ives, in 1756.

³ Ranald Martin's *Diseases of Tropical Climates*, second edition, p. 308.

⁴ Kenneth Mackinnon.

Grant in No. 9 of the 'Indian Annals of Medical Science,' there are two fatal cases of fever occurring in 1797, in which it is noticed that, late in the disease, 'the skin was tinged yellow' in one, and 'the skin was considerably yellow' in the other case. I see that both of these patients died on the eighth or ninth day of the fever. Henry Goodeve notices¹ the striking fact that, in the year 1770, *during a scarcity of rice*, nearly 2,000 Europeans, besides 80,000 natives, died of fever in Bengal alone. All these points combine in countenancing, to say the least, a strong suspicion that relapsing or famine fever occurred in Lower Bengal until late in last century. With regard to more recent times, Twining says, in 1835:² 'We every year meet with some patients in whom an intense yellow suffusion of the skin occurs in fevers of considerable severity.' When treating of that very grave disease which he termed the 'congestive fever of the cold season,' he says,³ 'Intense yellowness of the skin and eyes, with red-coloured urine, are occasional attendants on these cases'; and further on, in speaking of this fever as it occurred in natives, he says, 'Jaundice is by no means a rare sequel of the fever of this season.' Dr. John Macpherson informs me that, in a pamphlet which he wrote in 1856, he remarked: 'Cases do occur in which there is much yellow suffusion with a certain amount of black vomit, and such cases I have seen in Calcutta.' In the preface to his treatise, Dr. Lyons says: 'Since the manuscript of his work was transmitted to England in October 1871, I have personally observed two epidemics of relapsing fever in Bengal.' He appears to allude to the epidemic of dengue; but I think that we must await a fuller exposition of Dr. Lyons's views on this subject before we can attempt to argue the question further. I practised throughout the dengue epidemic of 1855 and during the first three months of that of 1871; and, with a clear recollection of most of my numerous cases, I am unable to think that I witnessed anything at all resembling true relapsing fever in either of those epidemics.

Treatment.—1. *Prophylaxis.* Removal of troops from impested localities, careful segregation of sick, good food, clothing, ventilation, and general hygiene, quinine and iron in tonic doses. As few prisoners as possible to be kept in jails. A famine was threatened in Behar when I was officiating as Inspector-General of Jails. I recommended to Government that summary punishment should be employed in all minor offences, that the jails might not become overcrowded.

2. *Treatment of the Disease.* The principal indications are to support life beyond the eighth day, and to counteract the tendency to sudden collapse by liquid nourishment, wine, and quinine; and then to watch the patient vigilantly, sustaining his powers and averting all injurious

¹ 'Sketch of the Progress of European Medicine in the East,' *C. M. T.*, vol. vii. part 2.

² *Diseases of Bengal*, vol. ii. p. 201.

³ *Ibid.* p. 349.

and depressing influences, until danger of relapse is past, and true convalescence is well advanced.

3. To give quinine steadily in moderate doses, first as the TEST [*vide* p. 83] of the true nature of the fever; and next, when we are certain that we have relapsing fever to deal with, as our most powerful nervine tonic and as an antidote to whatever paludal influence may be present in the system. To watch for and meet cerebral, thoracic, and abdominal complications. To prevent or relieve bowel-irritation, to which there is generally a great tendency, the exciting cause being often improper solid food eaten to appease the great hunger which is usually present late in the attack.

4. To aid the always slow convalescence by quinine, iron, and proper nourishment, and by change of air.

PERTUSSIS.

Mackinnon describes *Hooping Cough* as being epidemic in India (p. 128). In his experience, it was not extremely prevalent or aggravated in type in the plains of the North-Western Provinces. But, when he wrote, it was very severe at the hill stations, 'where,' he says, 'many deaths take place from it yearly among the European children.' This was written just as I went to India. I did not hear of the occurrence of such deaths in my time. He adds: 'It prevails as an epidemic all over the plains of the Ganges, usually in a very mild form, and most commonly showing itself at the same time of the year with the exanthemata so allied to it in many particulars.'

In a later communication¹ Mackinnon stated his opinion that the exanthemata and hooping cough are all known most commonly to show themselves at the change of season which ushers in the hot weather. He has sometimes seen the disease 'so mild as to render it difficult to distinguish it from common cough, although the manner in which the cough attacks, in fits more or less violent, and the specific hoop ought to be enough to guide us.' Noting the fact that an attack usually renders the individual exempt from future visitation, he observes that he has known children, who have had well-marked hooping cough in India, have the disease again severely in Europe. He again notices, that, at some of the Indian hill stations, the disease is severe and even fatal; which, he adds, seems to be another proof that, in the plains, it owes its generally mild character to the high range of temperature.

In a return, showing that the average strength for three years of European soldiers' children was 2,291, he found that there were only 23 cases of pertussis, being about eight per annum out of that strength. There was only one death.

¹ I. A., No. 3, for 1855, p. 178.

In his invaluable statistical tables, so frequently cited in these chapters, my friend, Surgeon-General Hugh Macpherson, shows that, during the four years ending 1853-54, there occurred, in the European regimental hospitals in the Bengal Presidency, six cases of whooping cough among soldiers, but no deaths. No cases of officers or women, 29 cases among children, or $\frac{3}{10}$ per cent. of strength, and two deaths or $6\frac{9}{10}$ per cent. of those treated. Dr. T. Farquhar reported that, at Mussoorie in 1862, there were forty-five cases of whooping cough and one death from pneumonia.

Among the 7,596 *children* of European soldiers in India in 1880 there were 104 admissions for whooping cough and three deaths. In 1881, there were admitted, in Bengal, 27 children, in Madras, 12, and in Bombay 28, of whom two died. In 1882, there were 22 admissions and one death in all India. In the three years only one of the large body of native prisoners was attacked—in 1881; in 1880, two Bengal Sepoys had whooping cough; and, also in 1881, a Bombay man was admitted. No other cases occurred in our native army in the three years.

Whooping cough prevailed from time to time in Calcutta. Few of the cases which I had were severe; but, when tracheotomy was recommended in pertussis, I opened, ineffectually, the trachea of an East Indian infant, at the moment when it ceased to breathe. A man in my ward was in considerable danger from pneumonic complication. I never saw phthisis set up by pertussis in India, and I did not hear of European children sent home to recover from its effects. Morehead says: 'In Dr. Coles's Report on Measles in the Byculla Schools, allusion is made to the presence of three cases of whooping cough at the same time. I do not find any account of the epidemic prevalence of this disease in these schools; but my impression is that it has occurred from time to time, during the last twenty years. In a note to the case of a child who died, as he considered, after recovery from whooping cough, Webb remarked: 'This is the only fatal case which I have met with in whooping cough in India. The disease has twice gone through the children of the Government Orphan School during the last four years, and once through La Martinière Institution.'

PAROTITIS.

Mumps is occasionally epidemic in India, the disease usually assuming the ordinary mild European type, and being unattended with severe fever or grave complications. But, on one occasion, I witnessed an unusual outbreak of this malady. There was, at Howrah, a single row of unhealthily situated detached houses, leading down to Tel Khal ke Ghat, principally inhabited by respectable Europeans. In front, separated only by the highway, ran an open, ill-dug earthen ditch, which was intended to carry the excreta of the large

native jail to the river Hooghly, but was practically an elongated reservoir of stagnant filth. Several European children in this row were attacked with mumps. The disease appeared to be endemic; I did not see or hear of any cases elsewhere. In most of the cases, the attack was mild and the children did well; but, in one of the houses, two fine little European boys became the subjects of acute membranous laryngitis, without the constitutional symptoms of diphtheria, when the parotid swelling was subsiding. There was no exudation about the fauces, but vomiting detached shreds from the air passage. The younger boy died suddenly while in the act of coughing. I found that the exudation tube, having become detached below, had been forced upwards and rolled together, and had completely filled and blocked the larynx. Had I been present when the accident occurred, laryngotomy would probably have saved him. The elder brother did well. The coincidence of diphtheria with mumps has been observed elsewhere. Morehead says¹ that cynanche parotidæa attacked the Byculla girls' school, Bombay, in March 1837. Seventy-four girls were affected, not a single boy. Mr. Carter reported that it broke out among the boys in October and November 1851. Seventy-five boys were affected, but only two girls.

The returns show that, in 1881, there were 23 admissions of European soldiers' children, for mumps, in all India. During that year the admissions of sepoys under this heading were 1,180. 785 prisoners had mumps. It is noteworthy that, in this year, sepoys were attacked in all the Presidencies, so also was the jail population, who are much isolated. There were some cases among the prisoners in British Burmah, and in the Andaman Islands. Twenty-one European soldiers were attacked; the European women escaped. Cases occurred among their children in each of the Presidencies. In 1882, 2 soldiers' wives and 14 of their children were admitted, and 901 sepoys and 769 prisoners had mumps, the disease occurring throughout British India and Burmah, but not in the Andamans.

In 1883, the statistics were:—European soldiers, 40; Children, 16; Women, none; Native soldiers in Bengal Proper, 419; Punjaub Field Force, 16; Central India Field Force, 27; Hyderabad Contingent, 9; Bombay Presidency, 409; Madras Presidency, 72; Prisoners, 473. It is mentioned in the report for this year that mumps had been prevalent throughout Assam for the past two years.

INFLUENZA.

Dr. John Adam placed on record² an account of what he termed an 'epidemic bronchitic fever' of infants and young children, which prevailed in Calcutta during the rains, or months of June, July, and

¹ *Op. cit.* p. 201.

² *C. M. T.*, vol. iv. p. 320.

August, 1828. This was preceded by a perhaps less general epidemic in 1824. In Dr. Adam's experience, not one child under four years of age out of a hundred escaped an attack. The victims were, for the most part, fine infants at the breast. Death did not occur in any child above two years old. The symptoms were those of capillary bronchitis, ushered in by a febrile paroxysm. In convalescence, there was a 'degree of weakness which was extremely protracted and difficult to remove.' This epidemic was not confined to the city and suburbs of Calcutta, but extended to many stations in Lower Bengal. It prevailed at Chinsurah, Burdwan and Bhauleah, and even as far northward as Maldah. In Calcutta, native children do not appear to have been attacked; but, at Burdwan, they suffered considerably. Here the adult natives had fever similar to that of the children, with the difference that 'it was accompanied by a troublesome ophthalmia.'

This epidemic appears to have about doubled the average death-rate of European infants in Calcutta. Three years later, in 1831, a remarkable outbreak of influenza occurred in Penang and Java. It was described by Dr. T. M. Ward¹ as 'the epidemic catarrh which prevailed at Penang in July and August, 1831.' Few of the inhabitants escaped a more or less severe attack. It affected at once whole families, attacking both sexes, young and old, of all races indiscriminately. It destroyed many old and infirm natives. There was fever with bronchitis. 'The depression of strength and spirits was considerable during the continuance of the febrile symptoms;' and, in convalescence, 'the patients were weak and languid.'

Again, three years later, in January, February, March and April, 1834, it was reported² that 'catarrh prevailed in Calcutta on so general a scale that it might be termed epidemic.' 'It attacked equally children and adults.' 'It ran its course through families with great severity.' 'In some cases, the affection of the head and larynx was very severe,' and, in others, pneumonia occurred; but there do not appear to have been any fatal cases. This was distinctly recognised as influenza. Two years and seven months later still occurred the great outbreak of influenza in England and France. Although I was a young student, I watched the progress and effects of the terrible influenza epidemic with which London was visited at the end of 1836 and in 1837, very fully and carefully, at Guy's Hospital. The main characteristics of this outbreak were debility and capillary bronchitis. Multitudes of aged people died; and, for several years afterwards, phthisical patients traced back their loss of health to the influenza of 1837. Webb, writing in 1848, speaks of the 'severe influenza of Bengal;' but Mackinnon³ says, with less than his usual cogency of argument, that in-

¹ *C. M. T.*, vol. vi. p. 125.

² *I. M. J.*, vol. i. pp. 67 and 146.

³ 'On the Epidemics of Bengal and the North-West Provinces,' *I. A.*, No. 5 p. 128.

fluenza had not been described by Indian authors, and that the disease was unknown in India. Assuredly there was not any great outbreak of influenza in that country in Mackinnon's or in my own time. I never saw anything of the kind there graver than a prevalent catarrh, neither has the United Kingdom been ravaged by this disease since 1837. Still I fully believe that, wherever catarrh or bronchitis runs through a town or a household, as they frequently do, both in India and at home, the malady is influenza, epidemic or endemic.

I am confident that any epidemiologist who will carefully review, in the originals, the narratives which I have cited, will feel convinced that an influenza wave extended from India to England between the years 1824 and 1837.

GLANDERS.

I saw a fatal case at Guy's, and offered to examine the body after death, but was not allowed to do so. I never saw this disease or *Farcy* in the human subject in India, although these maladies frequently attack studs in that country. A marked case of glanders, occurring in a native syce driver belonging to the 5th Battalion of Artillery, who was found to have had something to do with the veterinary department, and ten weeks previously had assisted in a *post-mortem* examination of a horse which died with a discharge from the nostrils, was described¹ in 1855 by Mr. Halls.

On May 2, 1885, it was reported in the newspapers that Mr. T. K. Cross, interrogated by Dr. Cameron, said that the Government of India had been asked by telegraph whether the 6th Bengal Cavalry had ever been free from glanders, or whether it had again broken out. This was the regiment of which one troop was glandered in Egypt during the campaign of 1882, and to which was imputed the infection of the 7th Dragoon Guards. The late Major Logan's death was not due to glanders, and no case of a native having contracted the disease could be traced.

The following case is narrated by Mr. E. F. Brockman:—A seaman, *æt.* 23, was admitted to the Madras General Hospital from a ship which had arrived from Melbourne with 200 horses. His duty had been to remove the excreta of the sick horses. His illness had begun six weeks previously by a severe sore throat, preceded by fever which was ushered in by rigors, and pain in the back and loins, followed by defluxion from the nostrils and the vesicular eruption. On admission, the body presented a large number of vesicles and bullæ, ranging in size from a pea to that of a turkey's egg. Some of these had burst, leaving large, raw, ulcerated surfaces, especially in the axilla, groin, scrotum, chest, back, and extremities. There was a

¹ *I. A.*, No. 4, p. 707.

discharge from the nostrils of a thick, adhesive, muco-purulent fluid tinged with blood. The dejecta consisted of an adhesive albuminoid fluid coloured with blood and bilious fecal matter. The margins of the eyelids were ulcerated, the cilia having fallen out. The tongue, lips, and teeth were covered with sordes. He lived three days. He stated that six horses had died on board from a disease which bore similar physical signs and symptoms.¹ A full account of the autopsy is given. Among the most important lesions were, a little blood coagulated on the mitral valve; tubercle-like deposits, each surrounded with considerable congestion, in the lungs; pulmonary cedema. The pulmonary mucous membrane throughout—larynx to the smaller bronchial tubes—were highly hyperæmic, and covered with a straw-coloured yellow membrane, removable by the forceps. Congestion and great softening down the whole tract of intestinal mucous membrane.

Malignant pustule.—I am told by my friend Dr. Pringle that he had under his care in India what he considered to be a case of malignant pustule in a person who slept next to a room in which hides were stored. All kinds of goat's hair, camel's hair, low-class Indian, Persian, and such-like wools are injurious to health.² Dr. Gordon notes³ that, in Japan, this disease, though occasionally seen in natives, is rare among foreigners, only one death from it being registered between 1871 and 1877.

Surgeon-Major J. North has reported⁴ the case of a Madras syce (horse-keeper), *æt.* 24, who, assisting in cutting up a horse which had died that day from anthrax, got accidentally an incised wound on the upper eyelid. Next day, the eyelid was inflamed, swollen, and closed, and there was brawny erysipelatous swelling of the whole of that cheek. Vesicles formed on the edges of the wound. Bursting, these left a dark sloughy surface with an indurated base. There was fever. The eschar, which occupied the whole eyelid, came away on the 22nd day. In 13 days the part was cicatrised.

For *Phagedæna*, vide *Paludal Cachexia*, *Scorbutus*, and *Hospital Gangrene*.

DIPHTHERIA AND CROUP.

He who would hope to throw a clear light upon the nature of the disease or diseases which, early in my career, used to be placed under these designations, ought to be an antiquarian as well as a pathologist. In this inquiry, it is to be asked, (1) How far these diseases used to be accurately differentiated forty years ago; and (2) Have changes since

¹ Mr. Sibthorp's catalogue of the Madras M. C. Museum, Prep. 38, Series 6.

² Aitken.

³ Chinese Reports, p. 197.

⁴ *J. M. G.*, January 1885, p. 1.

taken place in their type? I shall consider these points very briefly; but, having been in youth a victim to the neurosis laryngismus stridulus, or spurious croup, I may claim the questionable advantage of having some practical insight into this question—(1) It is certain that many observers, misled by the formidable character of the symptoms of false croup, have mistaken their cases of this disease for examples of the ‘membranous’ exudative type. An eminent authority on Indian disease wrote as follows, seven and thirty years ago: ‘Croup is a most frightful disease, attacking the strongest children and attended with the most agonising and convulsive efforts at respiration. It comes on quite suddenly, often in the middle of the night, and requires prompt attention and careful watching; for real inflammation of the larynx is apt to follow it. In common cases, however, all this hard barking clangor disappears after an emetic and a sleep.’ How came this generally very thoughtful pathologist to designate as ‘croup’ that which, by his own showing, was merely the ordinary type of laryngismus stridulus—spurious croup? Some years before his remarks were published, another Calcutta authority took a more just view of the case,¹ observing: ‘Spasmodic croup is a disease common to infancy from one part of India to the other, and not unfrequent at the Presidency’ (Calcutta). ‘Dr. H. H. Goodeve must have been often summoned from his couch to prescribe for this disorder, and to quiet the alarm of agitated parents. This, in appearance, is a very alarming disease, but is not dangerous unless neglected, and is very tractable.’ ‘The characteristic symptoms are—loud wheezing and difficulty in breathing. This disorder attacks suddenly, and generally between 11 at night and 2 o’clock in the morning, frequently precisely at midnight. The child starts from its sleep with the loud wheezing which may be heard throughout and without the house; we have known the sentry outside give the first alarm to the slumbering domestics. The child generally sits up in bed, and cries more from fright than pain or difficulty in breathing. There is a predisposition in some families to this complaint, every child being subject to it. Dentition predisposes to it. Disorder of the bowels acts sometimes as a remote and occasionally as a predisposing cause. It is common in India, from the Himalayas to the sea, though it seems to be more so in the Upper than in the Lower Provinces; and in some parts of these it occurs more frequently than in others. We would instance Rohilkund. During a residence of four years at Bareilly, we met with more cases than during an equal length of time in any other part of India. Its attacks are more confined to the cold season, or to the months ushering in the commencement and termination of this period of the year; we would fix upon October and March, when there is the greatest range of the thermometer in its diurnal changes, as the months in which it most frequently occurs;

¹ Finch’s *I. M. J.*, vol. ii. p. 428.

and, for this reason probably, the disease is more frequent in the Upper than in the Lower Provinces. We believe that, like inflammatory croup, it is often excited by a cold northerly or easterly wind, or by a wind blowing across a tank or river.' The writer adds that he knew of only one fatal case—a young infant died of convulsions, to which it was predisposed by a weakly constitution inherited from a delicate parent.

(2) Has any change taken place in the type of this class of diseases within the last forty years? I can declare positively, from my own personal and professional experience, that no material alteration has occurred in the type of spurious croup within that period. If membranous croup be diphtheria, its type may have undergone some change in Europe and in India; but I do not know that we have evidence of such change. We have always been told, until lately, that true croup attacks the healthiest children, and that it is essentially a 'sthenic' disease, attended with high fever. Mothers used not to be pleased when the healthy appearance of their young children was remarked. 'Yes,' it would be said, 'such children are very liable to croup!' In the first quotation given above, we see it emphatically stated that the disease 'attacks the strongest children.' Here it is to be borne in mind that, although I think it will be found that subjects of spurious croup are mostly rather pallid and deficient in muscular power, very fine-looking children suffer from this neurosis. Again, has there ever been a 'sthenic' type of diphtheria? I think not; but my experience in India—where the strongest Europeans have not always a very robust appearance—shows that many of the healthiest-looking people fall victims to diphtheria. A lady, who appeared to be in the enjoyment of the most perfect health, was attacked with diphtheria in Calcutta. I opened the trachea of a most beautiful and healthy-looking little European boy of four, almost *in articulo mortis* from diphtheria, whom one of my colleagues requested me to take charge of. At the Medical College, twelve years ago, the youngest and apparently the most vigorous of our staff succumbed to diphtheria. This disease may attack the strongest, but it is not, on that account 'sthenic'; further, those who, to ordinary observation, appear to be the most robust are often weak—*Nimum ne crede colori*. I am almost confidently inclined to believe that true croup has always been diphtheria of the air-passages, and that the natural destructiveness of the disease was, generation after generation, largely aggravated by heroic, depletory, and depressant measures, employed under the false idea that the malady is 'sthenic'; while many a little weakly but flushed-faced victim of spurious croup, who would have been asleep and well by sunrise had he been properly treated, has been bled, leeches, and blistered to death. Until treated, the subjects of spurious croup are generally feverish and flushed, but the skin acts freely after ipecacuanha and a warm bath. There is no

inflammatory fever;—no organic disease whatever throughout the progress of this neurosis.

Writing in 1860, Morehead did not allude to diphtheria or croup. Hence I infer that he had not seen the former and did not believe in the latter as a distinct entity. In his book, published four years later, Dr. Peet states that 'in the Western Presidency of India diphtheria in an epidemic form is quite unknown.' Peet 'never met with nor heard of croup in a native child in India,' and he held that 'it may, without hesitation, be affirmed that native children in Bombay,' and 'perhaps in other parts of tropical India, are quite exempt from this disease, whilst resident European children are frequently attacked.' I do not, however, infer confidently from his description that this authority was able to draw a clear line of differentiation between 'true sthenic membranous croup' and 'spurious croup.'

Mackinnon considered that croup is far less prevalent in India than in colder climates. Writing in 1855,¹ he cited an account of an outbreak of croup (?) which had just occurred at Darjeeling. During the first sixteen years of the occupation of that hill station as a sanitarium, croup had never shown itself, except on one previous occasion. The superintendent of Darjeeling called the outbreak 'the infantile epidemic,' and denied that it was a croup epidemic. Croupy symptoms did not appear at the beginning. There was 'an epidemic in which the mucous membrane of the fauces, throat, larynx, and trachea was affected' in various degrees of intensity,—from malignant sore throat, fatal in one boy of seven years, and croup with a false membrane, fatal in two cases under five years of age,—to the slightest coughs, colds, and sore throats, which were general among half the children of the place. Here, diphtheria and croup were, to say the least, simultaneously incident upon a small community. Total of children, say ninety. Here too, we cannot but observe the alliances of this expression of disease, on one side, to diphtheria, and, on the other, to the epidemic bronchitis of children in Lower Bengal, described under the heading of *Influenza*, p. 111.

I have seen many cases of diphtheria of the air-passages, and three marked cases of 'membranous' laryngitis; but I never saw a case of sthenic 'membranous croup' of the kind described in text-books of medicine some years ago. In very few of my cases was the 'brassy cough' present; but in all there was more or less of that harsh sound which is fairly represented by breathing hard and blowingly through the tube made by bringing the last phalanx of the thumb against the backs of the last phalanges of the fingers. The hill station of Nynee Tal long had an evil reputation for croup. Dr. Francis states, from three years' experience, that the children there were very liable, in the spring and autumn, to 'attacks of bronchitis, croup, influenza, and cynanche.'

¹ *I. A.*, No. 5, p. 131.

He observed that in 1855 '*croup of a sthenic character*' (the italics are mine) 'and bronchitis were epidemic at both the above seasons; whilst there only occurred two or three cases of simple cynanche tonsillaris. But, in September 1856, the cynanche cases were more numerous, and, in one family, they assumed a decided diphtheritic character. Here, again, we notice a close approximation of cases of 'true croup' (diphtheria of the air passages?) and of ordinary diphtheria.

I, as I have said, suffered much in childhood from neurotic spurious croup, and I saw a great deal of that disease among children in Bengal. One damp and stormy night, at a boarding-house in Calcutta, I was busy placing one of my own children, violently attacked with spurious croup, in a warm bath, when I was urgently called to see two children of an officer, in the next apartment, who were similarly attacked. The same hot-water kettles served for both families. I passed nearly the whole of one night at Bishop's College with a very fine boy, the son of a clergyman. The boy looked so healthy, and was so hot and flushed, and the cough had so loud a brassy clangor, that I, for some hours, doubted whether I had not, at length, met with a case of genuine sthenic membranous croup. Treatment, however, showed that it was merely the neurosis. We place the child in a warm bath, wrap it in a blanket, make it vomit with ipecacuanha, and give it a brisk purgative (in these cases, and almost in these alone, I gave calomel, which acts very kindly). Sleep generally follows vomiting; and, when the cathartic acts, the child is well, but rather weak.

Although nearly up to the time of my entrance to the Indian Service in 1848, I was an office-bearer at the Pathological and South London Medical Societies, and a steady worker at other societies, and visited Guy's Hospital almost daily, I knew very little indeed of diphtheria, except that which I learnt from French authorities on the *Diphthérie* of Bretonneau. I first saw typical cases of this disease in India. According to Kenneth Mackinnon, diphtheria 'seems to have first shown itself in Calcutta in 1852, when it destroyed several children at the Martinière School and elsewhere.'¹ Although this school is a very fine building, it stands almost at the extreme east of the city—a position which has always been regarded as unfavourable. The close vicinity of a very extensive and full Mohammedan burial ground was another evil, but Dr. Webb attributed the outbreak to the foul state of the drains, and to the presence of numerous sheep pens in the immediate vicinity.

Mackinnon says that it appeared again, in 1855, in Calcutta, but not in the same locality. He mentions that there had been one fatal case, a most sudden and afflicting visitation, occurring in a young married lady. He knew of another young lady, from whose fauces flakes of the

¹ 'On the Epidemics of Bengal and the North-West Provinces,' *I. A.*, No. 3, p. 176.

exudation were removed by the forceps. From that time, sporadic cases, with a distinct tendency to spread in families, have rather frequently occurred in Calcutta ; but, as far as my own observation extends, there has not been an epidemic outbreak in that city. It is, however, very noteworthy that, a year later, in 1856, diphtheria attacked four members of a family resident at Nynee Tal, a delightful hill station at an elevation of 6,450 feet above the level of the sea, upwards of 600 miles north-west of Calcutta.¹ Nearly every Indian writer on diphtheria moots the question, Is this disease closely allied to scarlatina ? Mackinnon states the barely noteworthy fact, that one of the Calcutta young ladies mentioned above as suffering from diphtheria in 1855, had a scarlet eruption, not preceded by fever, some weeks after recovering from the throat affection.

The late Dr. Peet, of Bombay, states² that, during a severe epidemic of scarlet fever, which raged in the southern part of Australia in the year 1853, diphtheria was very prevalent and fatal ; and, indeed, in some of the cases which he saw, it was difficult to say which of those diseases was present. Until scarlet fever was introduced into Australia, diphtheria had never occurred there. He adds that, in the Western Presidency of India, where scarlet fever did not prevail when he published his work in 1864, diphtheria in an epidemic form was quite unknown.

Dr. Francis witnessed an outbreak of diphtheria at Warley Barracks in 1857, scarlet fever being epidemic at the same time. He alludes to this after describing the four cases of diphtheria which he saw at Nynee Tal, where no suspicion of the existence of scarlatina existed. He suggests the question : Is diphtheria scarlatina without the eruption ?—a question which, I believe, most observers will answer in the negative. We have seen that, of late years, scarlatina has appeared in the Bombay and Madras Presidencies, having been imported in troopships ; but the fact that diphtheria has prevailed in Calcutta for upwards of thirty years, although no unimpeachable case of scarlatina has been observed in Lower Bengal, goes far to show that these two diseases are, to say the least, very separate entities.

The returns of our *British Force*, in all India, for 1880, show two deaths from diphtheria ; for 1881, only one death ; and for 1882, no death, in a force 57,269 strong. There was not any case of diphtheria among the *women* during the three years ; but, among the *children*, numbering from 6,208 to 7,596, there were, in 1880, 18 cases of diphtheria, with 11 deaths, and 40 cases of ‘croup and laryngitis,’ with 14 deaths. In 1881, diphtheria did not occur among the chil-

¹ Dr. Francis, ‘Diphtheria and the Malignant Sore Throat of Scarlatina,’ *I. A.*, No. 13, p. 9.

² *Principles and Practice of Medicine : designed chiefly for Students of Indian Medical Colleges*, p. 357.

dren, but there were 388 admissions for croup and laryngitis, and 28 deaths. In 1882, no mention is made of diphtheria, but 26 children were treated for croup and laryngitis, and 7 died. During the three years, only two admissions for diphtheria occurred in the *jail population*. In 1880, their average strength was 106,763, and, in that year, one case (fatal) was registered in the Bengal, and one in the Bombay Presidency. There was only one admission (in 1880) for diphtheria in the *Native Army* in the three years. In 1882, this force was 114,894 strong.

In my time, diphtheria was rather common in and out of Calcutta, and probably is so still. As I have already mentioned, one of our hospital surgeons fell a victim to the disease, contracted in examining a patient in his ward. I opened the trachea in two cases—in that of the European boy alluded to above, and of a young native man. My colleague, Mr. Partridge, requested that I would (my residence being in the college) watch the latter. He had come in much exhausted, thin rags of dark-coloured exudation matter, like sodden brown paper, hanging from his soft palate. As I stood at the foot of his bed looking at him, foam gathered at the corners of his lips, and in a moment I saw that he died. I did not lose an instant in making the first incision with my penknife, while I called for the scalpels and cannula, which were close at hand. A large tube was inserted, and Sylvesterism was immediately employed, and long maintained, but quite in vain. This case forcibly illustrated the almost instantaneousness with which death from exhaustion occurs in this disease. It was in strong contrast with that of a patient of Sir Joseph Fayrer's, who, having some considerable time previously had his trachea opened, suddenly pulled the cannula out and appeared to die immediately. A nurse ran out of the ward into the operating theatre, and called Sir Joseph, who was operating and could not leave. I went, at his request, put in the tube, and, as a matter of routine, but without any hope of saving the man, I employed Sylvesterism, and was astonished to find that breathing was restored. I published the diphtheria case some time ago, and think that a hint was made soon afterwards that I had forced the exudation down the trachea and so arrested respiration. This positively did not happen. I examined the case very carefully *post-mortem*. There were mere traces of thin exudation in the air-passages, but nothing that could occasion obstruction; and, as we worked the chest, mechanical respiration was perfectly free, air passing through the cannula at every stroke as if through the nozzle of a bellows. This was the only case of diphtheria which I saw in a native.

Under the head of *Aphthæ*, I shall allude to the fact that Anglo-Indian children and adults, returned from the East, often suffer severely from exudative aphthous inflammation of the soft palate and fauces, which, at first sight, suggests the idea of diphtheria, and which

has, I believe, been mistaken for that disease, much to the alarm and detriment of weak and nervous patients and their friends. I have seen a good deal of *Thrush* in patients, mostly aged, dying from exhausting diseases; the alliance of this condition, when fully developed, to diphtheria appears to be very close.

Nothing but the discovery of exudation confined to the air-passages of one whose symptoms I had observed to be those usually ascribed to true croup—not those of ordinary diphtheria—would convince me that I had seen a case of deadly croup. Never having seen such a case, I remain in a state of agnosticism on this important point; but, as I have already said, with a strong bias towards the opinion of those observers who regard ‘membranous croup’ as ‘diphtheria of the air-passages.’

CEREBRO-SPINAL FEVER.

There were, at my hospital, some three cases of what much resembled cerebro-spinal fever. In one, which occurred in my colleague's ward, there were, I understood, most of the leading characteristics of that fever. I had under my care a little boy who recovered. The symptoms were not unquestionable, but there was marked stiffness of the neck. There was no evidence of contagion or of the existence of an epidemic; there were not any maculæ. My friend, Dr. Joseph Ewart, who had then charge of the Calcutta General Hospital, did not see a case either at that time or during the whole period of his large Indian experience. I have recently made inquiry of three other eminent brother officers, whose long observation extended over the whole of the Bengal Presidency. Dr. Edward Goodeve and Dr. Theodore Duka watched sedulously for this disease, but never saw it in India. Dr. John Macpherson had one very suspicious case in an East Indian girl in Calcutta, but no more. The disease certainly belongs to India, and I was always upon the look-out for it, after my case occurred.

In his work on *Spirillum Fever* (p. 436), Dr. Vandyke Carter notes that four cases of ‘cerebro-spinal meningitis’ occurred in coincidence with an outbreak of the above-named fever at Bombay. He remarks: ‘I am not aware that this affection had been previously recognised in Bombay. It was, with typhus, an early attendant on the epidemic at St. Petersburg in 1864–5; and also at Breslau in 1879, again with ordinary typhus.’

The returns of the S. C. for 1880 give no case of this disease. In 1881, there was a fatal case in a European woman. Two sepoys and one prisoner recovered. In 1882, one European soldier and two prisoners were admitted and died. In the following year, a sepoy died, and there were five deaths among seven prisoners who were attacked.

ENTERIC FEVER.

The true enteric fever of Jenner is a disease of considerable antiquity in Europe. In 1576, Walter Devereux, Earl of Essex, Lord Deputy of Ireland, father of Elizabeth's favourite, died in Dublin of a common malady then known as the 'Irish Disease,' and afterwards called 'epidemic dysentery,' the modern Irish history of which has been given by Copland. This was manifestly a type of enteric fever. Camp and ship dysentery appear to have been the same disease, *plus scorbutus*.

Sir James Paget discovered 'specimens of typhoid ulcers, preserved by Hunter' [who died in 1793], in the College of Surgeons Museum. There is nothing to show that the *Dothinentérite*¹—of the separate identity of which the researches of Broussais, Petit, Bretonneau, and Andral gave evidence—was new to France when they investigated it; or that what Richard Bright called 'typhus with bowel complication' was of recent development in England, although it was long thought that fever attended with grave lesions in the follicles of the small intestine was more prevalent in France than in England; and Bright, in giving his first description of true enteric fever in 1827,² appears to have observed little or nothing of this disease before it came under his notice in the last quarter of the year 1826. Ten years later, when I attended his *clinique*, he was perfectly familiar with it, regarding the intestinal lesion as a complication of typhus.

The questions, *Is true enteric fever of old standing or of recent origin in India?* and, *To what extent does it now prevail in that country?* are of great importance to our professional brethren in the East.

I may first notice that Dr. Brinsley Nicholson stated in 1879,³ as the result of ten years' experience at the Cape of Good Hope and Natal, that he never saw or heard of a case of true typhoid. He thus confirmed the observation of Stewart Henderson, Apothecary to his Majesty's Forces at the Cape, who, writing in 1798,⁴ remarked that the Dutch inhabitants are exempt from many of those endemic and epidemic diseases which rage in other parts of the world. Henderson added: 'The small-pox, measles, remittent and intermittent fever, and that most fatal of all diseases—the jail, hospital, or ship fever—which

¹ The spelling *Dothientérie* is of recent adoption.

² 'Cases illustrative of the Morbid Appearances which occasionally take place in the Intestines during the Progress of Fever,' *Reports of Medical Cases*, 1827, p. 178.

³ *M. T.*, July 5, 1879, p. 5.

⁴ *Inquiry into the Causes which produce Disease among the Troops at the Cape of Good Hope, with a view of discovering the Most Effectual Means of Prevention.*

destroys so many of the human species in every part of Europe, are never generated here.'

On the other hand, I am informed by Inspector-General Lawson, that he has found evidence of the presence of typhoid at the Cape in 1827. Facts have recently been adduced by Surgeon-Major A. Clark,¹ by Surgeon-Major Black,² and by other observers, showing that true enteric fever prevailed rather extensively among our European troops during the Zulu campaign.

Whether enteric fever has long existed in Bengal, or whether it originated in my own time, is a question still open to debate. The writers on the diseases of India who preceded Annesley and Twining were, for the most part, ships' surgeons, who could only describe those maladies which came under their notice while they remained in port; and they might readily have overlooked the existence of a disease which, like true enteric fever, is decidedly rare in India. Still the works of these authors contain some noteworthy statements regarding Indian fevers with bowel complication. Thus, Dr. John Clark has given³ an interesting abstract of the narratives of fevers which occurred in the East India Company's ships from 1770 to 1785. (In the following quotations the italics are mine.) In the 'Triton,' after leaving Bengal, there occurred so much fever that the sick-list was increased to sixty when they arrived at the Cape of Good Hope. 'In the beginning of this fever,' the surgeon says, 'inflammatory symptoms chiefly prevailed, frequently with bilious vomiting; *but, in its progress, it changed into a typhus, or was succeeded by a flux.*' Remittent fever was very prevalent and fatal amongst the ships at Bengal after the rainy season of 1783. 'After the remittent, an ague was very frequent, in the middle of October, *which usually degenerated into a continued fever or dysentery, and in those forms proved mortal.*' In 1784, seven ships stationed at Kedgerie (at the mouth of the Calcutta River) lost, at that place, 170 men. 'The diseases which prevailed were remittents and intermittents and the dysentery, all generally attended with diseased viscera. A few died of remittents, *but the principal number of dysentery, which generally attacked those recovering from fevers.*' Mr. Lyons has argued, I think not conclusively,⁴ that Charles Curtis, surgeon of the 'Medea' frigate, described⁵ the true enteric of India under the designation of 'bilious fever and flux.' In a careful analysis of Sir James Annesley's descriptions of

¹ As cited in Sir J. Fayrer's Presidential Address for 1879 to the Epidemiological Society.

² *M. T.*, vol. ii. 1879, p. 554.

³ *Observations on the Diseases which prevail in Long Voyages to Hot Countries; particularly on those in the East Indies, &c.*, 1792, vol. ii. p. 464.

⁴ *I. A.*, No. 28, p. 254.

⁵ *An Account of the Diseases of India, as they appeared in the English Fleet and the Naval Hospital at Madras in 1782-3.* London, 1807.

the remittent and continued fevers of the Madras Presidency, contained in his great work, published in 1828, Mr. Lyons cites the following passage from Annesley's remarks on 'the organic changes in inter-tropical fevers,' as evidence that true enteric fever then occurred in that part of India: 'Marks of disease of the small and large intestines are generally confined to their internal tunics. The duodenum, jejunum, and ileum—especially the duodenum and termination of the ileum—very frequently are diseased in their mucous surface, which is inflamed in patches, sometimes covered with a muco-purulent secretion, and studded with small ulcerations, particularly the termination of the ileum. Occasionally the mucous surface is of a brick-red or purplish shade of colour, apparently ecchymosed, and covered with a bloody sanies, and readily detached from the subjacent textures. In several cases the ulcerations, which sometimes are large and far apart, at other times small and agglomerated—especially the former—have nearly penetrated the tunics of the intestines; and in a very few cases we have observed this occurrence actually to have supervened, the contents of the bowels being partly effused into the peritoneal cavity, and having produced peritonitis' (vol. ii. page 457).

Twining, writing on the fevers of Bengal in 1835, must have been well acquainted with true enteric fever, as the typhus with bowel complication of Richard Bright or the dothinentérie of Bretonneau, who had described it ten years previously, in 1825. Still he asserts, 'Typhus is rare in India.'¹ But he says nothing more regarding this rare Indian typhus until further on, in speaking of what he terms the congestive fever of the cold season (under which denomination he appears to have placed several distinct forms of disease), he observes,² 'In a few rare instances' (of a continued fever closely resembling the true enteric) 'where patients have died after a protracted fever of this sort, superficial ulcerations of the mucous membrane of the small intestines were found. I will not venture to assert that the ulcerations above alluded to ought to be considered as causes of the fever of the cold season; and my reason for not deeming that pathological condition a primary affection existing at an early period of the disease is that active purgatives may be repeated daily for a long time at the commencement of this fever without producing irritation. In fact, they almost always afford relief, whereas we do sometimes find that active purgatives produce a degree of irritation at a late period, and when a fatal termination takes place afterwards, ulcerations of the small intestines are found in these subjects.' I may interrupt this quotation by noticing that, at this point, Richard Bright would have remarked, as he has often done when I was his student, 'When you admit a

¹ *Clinical Illustrations of the More Important Diseases of Bengal; with the Result of an Inquiry into their Pathology and Treatment*, second edition, vol. ii. p. 201.

² *Ibid.* p. 353.

recent case of typhus, there is generally constipation, and it is needful that you should open the bowels; but in this disease they are irritable, and therefore you must give only two drachms of castor oil or you will convert this case of typhus into one of typhus with bowel complication' (by which he meant the true enteric of Jenner), 'which is a far more serious condition.' Twining continues: 'If more extended observations should prove that these ulcerations of the small intestine exist generally in the cases which terminate fatally, and that such a pathological condition is rarely met with in the inspection of subjects that have died of other descriptions of fever in Bengal, I should be inclined to adopt the opinion of Dr. Boot, that a peculiarity of the disease would be thus ascertained, which, combined with the exclusive prevalence of this fever in the cold season, its insidious invasion, obscure symptoms, slow progress, and protracted course, attended with prolonged stupor and delirium, and the organic changes at its latter stages, might establish a resemblance to some modifications of European typhus' (Yes, dothinentérite! Bretonneau would have said; Yes, typhus with bowel complication! Richard Bright would have said; Yes, true enteric fever! Jenner would have said—each of these four authorities calling the disease by the name by which he had distinguished it); 'although,' Twining adds, 'the resemblance be not strictly correct in all its details.'

After this it can scarcely be doubted that Twining met with true enteric fever in Bengal. This interesting fact was recognised by the clear discriminative judgment of Dr. Edward Goodeve, when he wrote upon this disease, twenty-four years later, in 1859.

As I shall show in the chapter on the Causes and Differentiation of the Remittent and True Enteric Fevers in India, Dr. Maclean saw this disease in 1838 at Madras and afterwards at Secunderabad and in China.

From this time, I do not find any very definite observations upon the occurrence of enteric fever in India until my friend Mr. Scriven wrote upon it in 1854,¹ giving notes of three cases of true enteric fever which he observed in Burmah in 1853. Mr. Scriven has given a more extended commentary upon this disease, as occurring in India, in the 'Indian Annals of Medical Science.'² To Mr. Scriven is undoubtedly due the credit of first showing that the enteric fever of India is the true enteric of Jenner. Dr. Joseph Ewart has given an important narrative of cases of this disease as observed in 1885 in native prisoners in the Ajmere Jail.³ Dr. Edward Goodeve has admirably described the disease as he observed it in Calcutta in 1858.⁴ He remarked that he felt confident that he had seen in the Bengal

¹ *M. T. G.*, January 28, 1854, p. 79.

² No. 8 for 1857, p. 511.

³ *I. A.*, No. 7 for 1856, p. 64.

⁴ *Clinical Lecture on Typhoid Fever*, No. 11, p. 141.

Presidency many cases of this disease. He was nearly equally confident that he had met with it among natives several times. When his lecture was delivered he had not had opportunity of making any post-mortem examinations.

Several other valuable contributions to the history of true enteric fever in India have subsequently appeared, among which special notice is due to papers by Mr. Lyons,¹ and by Mr. J. E. Moffatt,² and to several interesting papers and reports by Massey, O'Brien, Cleghorn, Greene, and others, which have more recently appeared in the 'Indian Medical Gazette,' October to February 1878-79. We have accounts of the disease as occurring in the *Bombay Presidency*—by Mr. Hanbury in H.M.'s 33rd Regiment, at Deesa, in 1859,³ and in Bombay by Dr. Peet.⁴ In the *Madras Presidency*—by Mr. Cornish,⁵ by Mr. Ranking,⁶ and by my friend Dr. C. A. Gordon, in his 'Report on Typhoid or Enteric Fever in Relation to British Troops in the Madras Command,' issued by the Madras Government in 1878.

Until the year 1870 enteric fever had not a place in the statistical returns of the diseases of British troops serving in India. Its admission to the returns is due to the publication of a paper, attached as an appendix to the Report of the Sanitary Commissioner with the Government of India on Age and Length of Service as affecting the sickness, mortality, and invaliding of the European Army, by the late Dr. Bryden, Statistical Officer with the Sanitary Commissioner. He argued that the disease is, and has long been, nearly everywhere prevalent among European troops in India, young soldiers being so especially liable to it that he believes it to be the principal cause of mortality among them. He attributes it to the direct and indirect influence of heat upon the young and unacclimatised. He subsequently showed that the disease had been found among native soldiers and prisoners in jail. Previous to 1870, any cases of this disease which may have occurred in the European army must have been placed under the heading of remittent or continued fever. It is shown by the author of an able review of Gordon's Report,⁷ that a distinct and independent place has been assigned to enteric fever in the statistical tables since 1871; and, during the six years 1871-76, 1,311 cases and 571 deaths⁸ have been registered under this designation in the army of India,

¹ 'Typhoid Fever discriminated from Remittent Fever, Enteritis, Pulmonary Consumption, Dysentery, and Cholera,' *I. A.*, No. 28 for 1871, p. 241.

² *Ibid.* No. 30 for 1873, p. 373.

³ *Bombay Medical and Physical Transactions*, second series, vol. vii. 1861, p. 144.

⁴ *Ibid.* vol. viii.

⁵ *Madras Quarterly Journal of Medical Science*, vol. iv. p. 65.

⁶ *Ibid.* p. 511.

⁷ *I. M. G.*, October 1, 1878.

⁸ On these statistics Parkes remarked, at p. 635 of the fourth edition of his *Hygiene*—'the immense mortality of enteric fever to cases admitted shows that many cases were only diagnosed after death.'

whose strength has averaged 58,670 annually. During the same six years, 57 cases and 31 deaths have been registered among women—averaging in annual strength 6,445.

Notwithstanding the apparent clearness of the above data, the question, Is enteric fever at present a common or rare disease in India? is—ready as its solution ought to be—certainly one of the most perplexing as it assuredly is one of the most practically momentous problems with which medical men in that country have to cope. It being a plain fact that if we, encountering a case of paludal remittent with bowel complication, insist upon calling it true enteric fever, and treat it as such, withholding that free and steady use of quinine which is the only remedy, that case will almost inevitably end in death.

Dr. Bryden assuredly did a good and very needful work in giving enteric fever a place in the register of cases; but, after admitting this, I must join Dr. Gordon and confess my unwillingness to admit that we have proof sufficient to convince us that 571 European soldiers of the Indian army died in six years of true enteric fever.

If this disease has long been common in India, it is a very extraordinary fact that several of our best and most careful observers should have met with so little of it, although they sought it diligently by the light of home experience. I have already given the results of Twining's observation. When Dr. Morehead published the first edition of his treatise, in 1856, he believed that this malady did not occur in India, although the type of disease in one of the cases which he recorded (No. 32—that of the hussar) closely resembled that of enteric fever—a fact which he clearly observed when, after describing this case, he added: 'While retaining this case in its original position, I must admit that recent inquiry may suggest that it was true typhoid, not adynamic remittent.' Indeed, in 1843, six years before Sir William Jenner established the distinction between enteric and typhus fever, Dr. Morehead, in his notes on the malarious fevers of the European General Hospital at Bombay,¹ drew attention to an occasional anatomical relation between some cases of remittent fever and some forms of European continued fever. His words are: 'A state of ulceration of the glands of Peyer at the end of the ileum also occurs in cases of typhoid remittent fever with gastro-enteric symptoms, just as it occurs in European continued fever with typhoid symptoms and similar local complication.' In the second edition of his work, and in his remarks upon the admitted existence of Indian enteric fever, in the India Office Sanitary Report for 1868, Dr. Morehead steadily maintains that this malady was long of rare occurrence in India. I was favoured, in 1879, with a most interesting communication from Dr. Morehead, in which he clearly showed that, had enteric fever been

¹ *Transactions of the Bombay Medical and Physical Society*, first series, vol. vi. p. 193.

common in Bombay during his long experience, he and other Indian observers could not possibly have overlooked it except in a very occasional instance. He could say for himself that he went to India familiar with the fact that there was a form of continued fever, described by Louis, with Peyerian disease; and he followed the discovery made by Jenner some fifteen years afterwards, that this is a fever distinct from typhus. In a prefatory note to No. 5 of the 'Bombay Medical and Physical Transactions,' in 1843, he explained that it was his constant practice to examine all the organs, not only those expected to be diseased, and *specially to open the entire tract of the intestinal canal*. Consequently, he could not have overlooked the lesions characteristic of enteric fever had they been of frequent occurrence. Much of this article was written before I received Dr. Morehead's letter. It will be seen that I have held the same argument with regard to Twining and his Indian successors:—few of us would have overlooked cases of a disease with which home practice had made us perfectly familiar.

I had, in 1879, the advantage of a conversation on this subject with a senior brother officer who had recently completed upwards of thirty years' service, chiefly in the North-West Provinces. Throughout all that time, he had made the study of medicine a labour of love. He was secretary to the first Sanitary Commissioner with the Government of India, and had lately accomplished his five years' tour as Deputy Surgeon-General of one of the largest circles of inspection. Still it had never occurred to him to meet with a case of Indian enteric fever. He told me that Surgeon-General Irving, who was for many years Civil Surgeon of Allahabad, never met with a case until 1871, when he had to treat two cases in a civilian's family.

My friend Brigade-Surgeon William Curran is well known as having made Indian fevers a special study. During fifteen years he worked in European military hospitals and large stations in nearly every part of India. Still he only saw one case of true enteric fever, which occurred at the hill station of Murree.

Dr. Joseph Ewart's Descriptive Catalogue of the Pathological Preparations in the Museum of the Medical College, Calcutta, was published in 1865. That noble pathological collection then contained only six preparations illustrative of disease of the follicles of the ileum in true enteric fever. Two of these were presented by Mr. Scriven, and one by Dr. Eatwell. The others may have been Dr. Edward Goodeve's. I sent them two or three preparations in the following year (1866). Goodeve's, Eatwell's, and my own preparations, numbering altogether only nine at most, may be taken as representing all the fatal cases of true enteric fever which occurred in the physicians' wards, containing 120 beds, of the largest hospital in India, from 1858, when Dr. Goodeve had cases in the hospital, until 1876, when I ceased to be connected with the institution.

The following data, of separate periods, fairly represent the recent statistics of enteric fever in the European army in India. In his presidential address at the Epidemiological Society in 1879, Sir J. Fayrer mentioned that the Sanitary Commissioner's Report of 1877 shows that, out of 233 cases of typhoid, 92, or 39 per cent., proved fatal, the admission-rate being 4.1 per 1,000 of strength. It appears that 2.45 per cent. occurred at or under 24 years of age; 1.55 at 25 to 29; 0.99 at 30 to 34, and few or none above that age; showing that the disease tells most severely on the younger men—in this respect resembling typhoid in England. Again, Dr. Bryden, in his report of the Statistical History of the European Army in India up to 1876, said: 'It' [enteric fever] 'has no geography; and it is a matter of popular observation that no regiment or battery escapes enteric fever *in the first year*, whatever cantonment of India may be selected. Out of seventy-three bodies of men, two regiments and seven batteries only returned no case of enteric fever in the first year.' Bryden gave the following analysis of 368 deaths that occurred between 1823 and 1876:—

Ages								Total Deaths
24 and under	.	.	.	.	.	.	.	255
25 to 29	.	.	.	.	.	.	.	90
30 to 34	.	.	.	.	.	.	.	17
35 to 39	.	.	.	.	.	.	.	4
40 and upwards	.	.	.	.	.	.	.	2

Seventy-five of these deaths occurred within three years after landing in India, and 94 per cent. of the total were among men under 30 years of age. In a memorandum which Sir J. Fayrer had lately received, Dr. Bryden wrote that, out of 132 deaths from enteric fever in 1878, 90 occurred in men who had been under 22 months in India.

In his report on the health of the European army in 1883 the Sanitary Commissioner observes: 'The tendency to return an increasing number of continued fever cases as enteric fever has been frequently noted in former reports. The results of the year under review show that tendency still continued. In the army of India, 429 cases of fever were recorded as cases of enteric fever against 357 in 1882. These numbers yield ratios of 7.7 per mille of admissions in 1883, and 6.2 per mille for 1882. During 1883, 133 died of the disease, or 2.40 per mille against 2.55 in 1882. Notwithstanding the greater frequency of the disease during 1883 as compared with 1882, the mortality was therefore less. From 1870 to 1877 the admission-rate fluctuated between 2.8 and 4.6; and, during this time, the mortality per cent. of those attacked varied from 50.88 to 39.49, or a mean of 43.73. In 1878, the admission-rate rose to 8.5 per mille, or almost double the highest rate recorded during 1870 to 1877, and continued high during 1879 (8.0) and 1880 (7.9); the death-rate per cent. of those attacked remained much the same as it was during the period 1870-77, for it varied from

43·01 to 46·17, or a mean of 44·72. During 1881, the admission-rate again fell suddenly to 5·6; but the percentage of deaths somewhat increased (47·26). During 1883 the admission-rate again rose to a high ratio (7·7), but the death percentage was only 31·00, the lowest on record since 1870.'

Further on, the Commissioner observes that the figures for 1883 show that both the mortality from enteric fever and the ratio of liability to it are by far the greatest among young soldiers under 25 years of age. The mortality among these was 4·34; whilst, for men between 25 and 29 and from 30 upwards, it was only 1·50 and 0·70 respectively. The percentage of liability, among young soldiers under 25 years, was 66·36, the highest on record since 1877, and 10·8 per cent. higher than it was in 1882. The percentage of liability rapidly falls with increasing age. For men between 25 and 29 it was 22·94, and, for men above that, only 10·70. Full tables are given (page 28) in illustration of the law that this disease attacks soldiers of short residence in India in much greater proportion than others; and, further, that the greater the proportion of young unacclimatised men in the army of short residence in India, the greater is the liability of the army to loss from enteric fever.

One of the latest of Dr. Kenneth Mackinnon's admirable writings on Indian fevers was published in 1855,¹ when true enteric fever was beginning to be noticed in Burmah by Scriven, and in Ajmere by Ewart. Still, it is clear that this very accurate observer did not suspect that any of his own cases of remittent or continued fever were, in reality, examples of true enteric. But, in the following passage, he clearly recognises the observation of others in this direction: 'The congestive fever of the cold weather, described by Mr. Martin and Dr. Twining as prevailing in Calcutta among all classes, and the symptoms of which resemble so much the typhus of Europe, I have not seen to prevail generally, though I have of course met with cases where, with typhoid symptoms, the remissions have been so little marked that the case might be called continued.' He then notices that the Calcutta authors do not even hint at contagion.²

My own experience of true enteric fever in Bengal, in an extensive field of observation, diligently worked for upwards of twenty-seven years, was small, but very distinct. I went to India with a full practical knowledge, acquired in the wards and post-mortem room at Guy's, of that which I first knew as 'typhus with bowel complication,' or dothinentérite, and which I at once perceived, upon the publication of Jenner's views, to be his enteric fever. But I only saw, in the ten years, ending 1876, two distinct outbreaks of this disease, between which an occasional case occurred in my wards and practice at distant intervals. Throughout this decade, I and my house-physicians were constantly

¹ *I. A.*, No. 5.

² *Treatise*, pp. 247-48.

upon the look-out for enteric fever, examining the skin, taking records of the range of temperature, carefully inspecting the stools, and investigating the state of the intestinal follicles, in all fatal cases of a suspicious nature. All my cases occurred in Europeans and East Indians. In these two outbreaks, I had a sufficient number of fatal cases and post-mortem examinations to enable me to trace all the ordinary changes in Peyer's follicles, from simple infiltration with surrounding hyperæmia to fatal perforation.¹

I have no desire to attempt to prove that true enteric fever is not now as prevalent among European troops serving in India as Dr. Bryden and others have believed it to be. But the above facts tend to show that, if the disease is upon the increase in that country, its advance has been slow and its prevalence, among the civil population, has not been great. Still, for several years before I left Calcutta, in 1876, the general impression in the profession there was that cases of enteric fever were becoming more and more frequent, coincidently with the extension of a great system of underground sewage in the city. Nevertheless, I think that Dr. Gordon has done a very needful service to science by insisting upon a most scrupulous sifting of cases.

I think that a minute inquiry into the *prevalence of enteric fever among the Natives of India* is still a desideratum. In 1883, when the average strength of our native troops was 114,830, the returns show only seventeen admissions, with five deaths. Among the prisoners, numbering 88,174, there was only one case which recovered. In 1884 the native soldiers and prisoners had the same admission (·2) and death (·09) rates per mille.

Since Dr. Ewart wrote, in 1855, on the existence of true enteric fever among the native prisoners in the Ajmere jail, it has been shown by Bryden and others that the disease occurs in natives of India. The general consent of the profession appears to be that such cases are very rare. Until I left India, I used to hold rather warm arguments on this point with one of my native medical friends, he declaring that he had seen many hundred cases of enteric fever in Calcutta natives, I maintaining that I had never met with one, and challenging him to show me one. There are no post-mortem examinations in native private practice; and, in a considerable number of cases of remittent and intermittent fever, diarrhœa occurs vicariously, or dysentery sets

¹ This extreme lesion occurred once in the person of a young European sailor, who, when apparently almost recovered from the fever, became so voraciously hungry that, managing to obtain a new loaf unknown to the nurse, he tore it in pieces and rapidly swallowed the whole of it. He was almost immediately afterwards attacked with the gravest symptoms of peritoneal fecal effusion. I noted, at the time, that, although I would not altogether condemn, until I had seen it tried by anyone who would venture to try it, the late Dr. Samuel Denny Turney's practice (*Practitioner*, vol. xix. p. 337) of giving solid food in enteric fever, this by no means solitary case appears to bring strong argument against it.

in as a complication, where sweating is checked, especially in the cold weather and rains. Here there are not the follicular lesions characteristic of true enteric fever.

In a valuable paper, which Dr. Furnell published in 1879,¹ on true enteric fever in Madras he corroborates my opinion that, hitherto, this fever has occurred very distinctly but not very frequently among the civil population of India. He wrote: 'We have quite, in Madras (I mean the town proper), made up our minds not only to its existence, but to its being occasionally anything but rare. During the cold season of 1878-79, it was very common; and I had, in my wards, several times, as many as half a dozen cases together. I was sceptical at one time of its common occurrence, but last year quite removed my doubts.' Dr. Furnell gave one typical case in a Hindoo. He says that, previous to this, he had not seen any cases in natives, or he had overlooked them. In like manner, Dr. Porter, of the Madras Medical Department, stated² that reported cases of undoubted typhoid among natives are rare.

In 1879, I had a very interesting conversation with a practitioner of great experience in Ceylon, who informed me that true enteric fever was of such frequent occurrence in the *pettah* (native town) of Colombo, that he had of late found it very difficult to convince his younger medical brethren that some of their cases were examples of malarious remittent, and must be treated as such.

There has, for many years past, been a section of observers who have ascribed the occurrence of the ulceration of the small intestines, found in Indian fevers, to what they hold to be paludal remittent with bowel complication—not true enteric fever.

Thus Sir Ranald Martin noticed that, in very severe or very protracted cases of remittent, redness, ecchymosis, or even ulceration will be found on the surface of the large and small intestines. Dr. John Macpherson remarked³ that, in some fatal cases of fever which occurred in the hot weather of 1848 in the Calcutta Presidency General Hospital, Dr. T. M. Lee recognised the post-mortem appearances as being those peculiar to enteric fever. Dr. Macpherson, however, considers that these were cases of 'acute remittent fever,' and adds: 'Affections of Peyer's glands may take place in Bengal fever without their being necessarily cases of typhoid.'

In the second edition of his work, Dr. Morehead insisted that disease of Peyer's glands, either in the stage of turgescence or ulceration, is not a morbid state peculiar to enteric fever. It occurs in cholera, in protracted diarrhœa, in acute muco-enteritis, and as an occasional complication of remittent fever; he had also observed it in one or two cases of measles.

¹ *M. T.*, p. 631, vol. ii, 1879.

² *J. M. G.*, April 1, 1878.

³ *Lancet*, January 21, 1881.

In 1879, my late honoured colleague, Dr. Edward Goodeve, wrote to me that, although the question of drawing a distinct line between Indian remittent and enteric fever had occupied his attention, he considered that it demands further research. He suggested a patient sifting of the whole question, and especially an inquiry as to how far jungle fever resembles the enteric. Marked cases should be taken for comparison. He urged a re-investigation of jungle fevers and of those grave remittents which prevail in the vicinity of the Godavery, by the light of our present knowledge of the true enteric fever of India. It was long before Louis and Jenner established the distinct individuality of true typhoid, and we must not, he considered, be surprised at the existing difficulty and delay in differentiating these two fevers. He met with enteric fever at Cawnpore upwards of thirty years ago, and he always believed that there was a difference between it and remittent fever, especially in the manner of its progress, as compared with the quick course taken by most cases of remittent, which disease is remarkably frequent and severe in malarious seasons, and generally is more acute in its character and more rapid in its course than is the true enteric. In remittents, there are often evidences of malarious changes in the blood, as acute disintegration and leucocythæmia. In many remittents, he had found lesions of Peyer's patches closely resembling those in enteric fever, but it would be going too far to presume that the two sets of lesions are identical. He did not see the rash in India.

In 1879, I received another very interesting communication from my friend, Dr. Joseph Ewart, in which he states that, after he had observed his published cases at Ajmeer, in 1855, he, when attached to the Meywar Bheel Corps, recorded several well-marked cases among the Bheels of the regiment and the aborigines of the district, but native prejudice prevented him from verifying diagnosis by post-mortem examination. However, he is quite convinced that typhoid fever existed both in the regiment and in the surrounding villages, especially in the autumn and in the early months of winter, when the worst forms of malarious remittent abounded. This experience led him to inquire, in the 'Indian Lancet,' whether many of the malarious remittents were not, in reality, typhus, typhoid, or relapsing fever, modified and often materially aggravated by the presence of malaria. That this is so—that many of the so-called malarious remittents are simply typhoid or typhus fever modified, often masked, by malaria and malarious fever—he has repeatedly observed, ever since this declaration was made, both in and around Calcutta. He holds that, in a large number of so-called remittents with diarrhoea and severe typhoid symptoms, the chief factor is the typhoid fever poison; but when, superadded to this, the blood is surcharged also with a large dose of malaria, death often ensues from the combined operation of these causes before ulceration of the agminated glands occurs. This happened

in three of his patients, residing in the suburbs of Calcutta, in 1875, who succumbed in from eight to twelve days from what would formerly have been designated remittent or jungle fever; but in all there was very marked prominence of the agminated glands from acute specific inflammation without ulceration. Had the patients lived long enough, ulceration would have been duly developed. In both natives and Europeans who have been subjected to the influence of malaria, the fluctuations of temperature in enteric fever are greater than in those who have never been exposed to this poison, the morning remissions being so marked as to delude the observer into the belief that he is dealing with malarious remittent simply, when he has really to encounter true typhoid fever modified by the manifestations of infection of the system with marsh poison. In his practice at the Medical College and General Hospitals, and among the community of Calcutta, Dr. Ewart has seen the disease in all its phases—so mild that patients have gone about their ordinary business during the greater part of the fever, so severe as to end fatally in a few days, either singly from an overpowering dose of the poison, or when complicated with serious malarious poisoning; or to terminate by uncontrollable hæmorrhage from ulcerated agminated glands, or by perforation of the gut. In natives, the rash is not so easily made out as in Europeans; but, with care, he often distinguished it in those whose shades of colour verge upon brown rather than upon black. In a certain proportion of Europeans the rash is not observed. In fatal cases of this kind, he has found ulceration of Peyer's patches. Ever since attention was drawn to this disease by Mr. Scriven, who was attached to the General Hospital of Calcutta for many years, typhoid fever has been recognised as a constant factor in the sickness and mortality in that institution. During a long series of years, the conduct of the disease has been most carefully watched and fatal cases tested by post-mortem examination. The following statistical data are taken from Dr. Ewart's reports for 1873 and 1874:—

	1865.	'66.	'67.	'68.	'69.	'70.	'71.	'72.	'73.	'74.
Cases of typhoid fever treated }	17	18	12	5	4	14	15	17	7	11
Died . . .	3	3	4	3	3	1	5	11	3	5

Sir Joseph Fayrer holds the opinion that, in India, and probably in other hot and malarious climates, true enteric fever arises not only from pythogenic causes, but also very frequently—nay, perhaps more often—from climatic influences. This view tends to explain much of the difference of opinion which arose in the Gordon controversy.

If there be two forms of Indian enteric fever, there may, possibly, be two sets of intestinal lesions. My own English and Indian experience, however, shows no change in these lesions—those which I used

to find at Guy's in 1836 were identical with those which I found in Calcutta between 1866 and 1876.

It is clear that various degrees of hyperæmia of mucous membrane and considerable enlargement of the agminated and solitary follicles attend non-specific irritation of the small intestines. In a case where death occurred while a dose of elaterium was acting, I found the mucous membrane of the lower part of the small intestine highly œdematous and the Peyerian follicles much enlarged. There are several points of resemblance between Asiatic cholera and true enteric fever, especially in the fact that, in cholera, the intestinal lesions are generally most strikingly developed at the lower part of the ileum. But the follicular lesion in enteric fever is as characteristically and unmistakably specific as the vaccine vesicle is; and I consider that any case of protracted fever in which the lesions generally recognised as the tokens of true enteric fever are found to be present must be accepted as a case of that disease by virtue of the existence of these morbid changes.

For a considerable time past, there has been a growing tendency among the observers of Indian diseases to deny the existence of what I cannot but believe to be one of the most certain and indisputable entities in nature—the paludal poison. My able friend, Dr. C. F. Oldham, and some American authorities, declare that ‘malaria, as a specific poison, does not exist.’¹ After twelve months’ experience at the Calcutta General Hospital (where, by-the-by, paludal remittent is not at all frequent among the European patients, although there is abundance of it among the native poor at the Medical College Hospital, two miles off), Mr. Lyons exclaims, ‘I am disposed to believe that remittent fever is a myth, like malaria, the fever-producing principle which is said to produce remittent fever.’ And again: ‘It appears to me that, at present, remittent fever is a theoretical synthesis in this country of the continued fevers of Great Britain, and should be discarded as an unscientific entity.’²

Dr. Morehead long ago foresaw the confusion which would result unless the most scrupulous accuracy was brought to the differentiation of the enteric and remittent fevers of India; and it is to be feared that, for some time longer, we shall hear of such differences of opinion with regard to type, and, what is of much more importance, with regard to treatment, as arose a few years ago between Dr. Gordon and his officers in the Madras Presidency—he believing in marsh malaria, typhus, and enteric fever, exclusively of pythogenic origin, and also in paludal remittents; they viewing all the fevers of India, with the exception of intermittents, as ‘the continued fevers of Great Britain,’ and probably

¹ *What is Malaria? and Why is it most Intense in Hot Climates? An Inquiry into the Nature and Cause of the so-called Marsh Poison.* Preface, p. 6.

² *Loc. citat.* pp. 269–70.

being more or less sceptical with regard to the existence of a marsh poison. I am able to listen with real interest and respect to the arguments of Messrs. Oldham and Lyons;¹ but I cannot abandon the belief, founded upon the observation of a lifetime, that there occur every year in Bengal thousands of cases of what I recognise, by its history and symptoms, as paludal remittent, with or without bowel complication, more or less amenable to quinine; and that there also occur, only at intervals, small groups and isolated cases of that which, also by its history and characteristics, I perceive to be enteric fever—a disease which it is as impossible to arrest by quinine as it would be to batter down the Tower of London with feather bolsters.

I fully grant, with Dr. Ewart, that true enteric fever may be so complicated with and masked by the marsh poison as closely to resemble, and often to be mistaken for, paludal remittent. Still, against the arguments of Mr. Lyons and others, I maintain that no two entities in nature are more essentially distinct from one another than are the marsh remittent fever and the true enteric fever of India.

In discussing a case which they freely admit presents in all other respects the typical characteristics of true enteric fever, Dr. Gordon and other Indian observers appear to have been met by the objection—Can this case be distinctly traced to the direct effects of filth-poisoning? If not, we cannot accept it as an example of true pythogenic enteric fever. Upon this point, although I am clearly of opinion that filth only acts as an EXCITING cause of true enteric fever, I must say that, in all probability, there is no country in the world in which impregnation of the soil with cesspool filth is so general as it is nearly throughout the plains of India. I had, many years ago, a correspondence with the late Colonel James, then executive engineer of Berhampore, at the end of which he told me that my prescription for the cure of the great tank attached to the European barracks at that station had been successful. Marvellous to relate, the barrack drains were led into this otherwise fine sheet of water, out of which the soldiers used to drink! A brother-officer told me that he saw the water black with bubbles of noxious gas floating upon its surface. It is not surprising that these stately and most costly barracks were, after many years of terrible mortality, declared to be untenable by troops. The remedy consisted mainly in covering the bottom with a thick layer of pure earth, in planting vallisneria, and by largely stocking the water with fish.

In writing on the sanitary prospects of Calcutta, I once had occa-

¹ Except when the latter (probably by a *lapsus penne*) says, at p. 277, that Dr. Chevers has pointed out to him that hæmorrhagic dysentery 'is probably typhoid fever'—I having, in writing to him, advocated the opinion that the ship-dysentery, described by Lind and Trotter, was probably enteric fever in scorbutic subjects. Nothing can, demonstrably, stand farther apart from enteric fever than does the hæmorrhagic dysentery (sloughing colitis) of Bengal and other districts of India.

sion to inquire: Given a loaf of bread that has been thoroughly saturated with sewer-filth, how is it to be purified; and what means, within reach of our science, are adequate to cleanse the loose soil of this city, which has thus soddened for two hundred years? The rural inhabitants of Bengal frequently change the locality of their dwellings. I have travelled over nearly the whole of the plain country of the Delta, and I believe that there are very few eligible localities which have not, at some time or other, been occupied by huts. Dr. William Palmer tells me that he has been particularly struck with this point, in its bearing upon the causation of enteric fever, when engaged in the examination of potable waters in his capacity of chemical examiner to Government. He very rarely found a Bengal water free from traces of contamination by animal filth. The Calcutta Midaun, a large grassy plain between the city and Fort William, is generally supposed to have been originally rice-fields. An apparently unexceptionable site for a new tank having been selected there, in what was imagined to be virgin soil, Dr. Palmer found the condition of the water to be such as to lead to the discovery that a village had formerly occupied that spot. I think that it may be fairly inquired, Is there any bazaar in India, accessible to European soldiers, in which a thirsty man is not likely to obtain a draught of filth-impregnated water? Nay, more, is there any such place, out of the Presidency towns (where the municipal water supplies are good), in which there is the slightest hope of obtaining a draught of perfectly unexceptionable water?

The practical outcome of Bryden's researches into the prevalence of enteric fever among European soldiers, in India, and of the Gordon controversy as to whether the majority of the cases now reported by medical officers as enteric fevers are, in reality, examples of that disease, or cases of paludal remittent with bowel complication, may be embodied in this brief advice to young medical officers:—Let the practitioner approach each of his fever cases, where the disease assumes a continued form, in a spirit of vigilant inquiry, and never rest satisfied until he has sufficient grounds for determining whether it is one of remittent or of true enteric.

The remittent is, if we can only take it early enough in a good constitution, curable by quinine; the true enteric is not, but is gradually benefited by it. The temperature charts of the two diseases are altogether dissimilar. At the onset of paludal remittent, before quinine begins to act, the highest temperature is immediately attained. In true enteric fever the rise is slowly gradual. The condition of the intestinal follicles should be carefully examined in every fatal case of fever; the lesions of true enteric fever are, as far as we know at present, quite unmistakable. The body of every European soldier dying in hospital in India is examined post-mortem. If undeniably characteristic follicular changes are discovered in nearly a hundred cases

annually, the reports might easily be tabulated by the British Medical Department and published for general information. In this inquiry we are guided safely by closely observing the action of quinine, especially noticing its influence on the temperature. The *quinine test* (p. 83) must be employed fairly in every case. The practitioner should never allow his over-confidence that the disease is true enteric fever to deprive his patient of a fair trial of quinine treatment.

It would be well if, for several years to come, all candidates for Indian appointments should be expected to produce certificates of having carefully studied true enteric fever in wards and post-mortem room. And every one who goes to India should be supplied with a card of accurate coloured representations of all the morbid changes to which the follicles of the ileum are liable.

Preventive measures.—We have before us the suggestive fact that very young soldiers, when conveyed by the most rapid means of transit, suffer soon after arriving in India or in South Africa, or, as dire experience has now shown us, in Egypt, from true enteric fever, a disease which appears to be rare among the native and civil European inhabitants of those countries. Consequently, it is needful (1) that, as Sir J. Fayrer long ago pointed out, in selecting troops for service there, the men's age should be well considered; and (2) that a company or two of soldiers should be sent to India in slow-sailing vessels by the old route round the Cape, and that it should be most carefully observed whether true enteric fever occurs among them either during the voyage out or within the first six months of their residence in India.

In 1879, the editor of the 'Medical Times and Gazette' published an important leader,¹ in which he reviewed Surgeon-General Gordon's report, and another report by Surgeon-General J. Kerr-Innes, on the prevalence and causation of typhoid fever among European troops in the Bengal Command in 1876, as they have been considered by the Army Sanitary Commission. The latter authorities conclude their report by recommending (1) that, if practicable, soldiers under twenty-four years of age should not be sent to India, or that young soldiers be sent to hill stations as soon as they arrive; (2) that strict conservancy of bazaars and towns frequented by the troops should be rigidly enforced; (3) that sources of malaria in stations and barracks should be detected and remedied; (4) that existing unwholesome conditions of stables, barrack-room floors, and adjacent sub-soil drainage, should be immediately remedied; (5) that greater care appears necessary in protecting drinking-water from pollution. They insist upon fuller, and more minute and careful records of all cases of fever; diagnosis being confirmed by faithful, exact, and detailed reports, giving the history, the surroundings, all the symptoms and signs, including the temperature from day to day; and, in fatal cases, the post-mortem appearances.

¹ *M. T. G.*, September 6, p. 265.

I have nothing original to suggest with regard to the treatment of true enteric fever in India. Here, as in most other Indian diseases, we must not overlook the presence of the malarial taint. I gave quinine, seldom ordering doses of more than four grains, throughout the attack. I never imagined that quinine would arrest the disease, but it always appeared, beyond any other agent with which I am acquainted, to moderate the temperature and to maintain vital power. Quinine does not cure Indian enteric fever, but it often carries the patient through; and I believe that, without it, the recoveries would be few or none.¹ I always gave wine and brandy freely, but I have never been in accord with those who believe that, in this disease, alcohol is more valid than food. He was, as nearly all who have treated enteric fever and typhus agree, a sound physician who desired that the words 'He fed fevers' should be inscribed on his tomb; but, in this fever, quinine and alcohol are, I consider, only valid when they do no more than enable the system to receive adequate nourishment during all those weeks of inevitable trial, and of waste and degeneration of tissue. I am unable to follow the reasoning of those who, in this disease, are not satisfied with employing alcohol as a means of sustentation, but carry it forward to the point of over-stimulation.

For additional remarks on Enteric Fever, see Malarial Remittent Fever.

YELLOW FEVER.

In the chapter on Indian Relapsing Fever, I mention that, during the Burmese war in 1852, cases of fever occurred among H.M.'s troops, especially in the 80th regiment, which, in the opinion of the medical officers who were acquainted with yellow fever in its natural habitat, exhibited apparently a perfect identity with the yellow fever of the West Indies and the bilious remittent fever of Sierra Leone.² This may have been either Indian relapsing fever with yellow suffusion, or a grave form of malarious remittent attended with blood-disintegration. In protracted campaigns in India, and especially in *Burmah*—a fact of great importance at the present time—the presence of a scorbutic taint has generally been recognised, and is always to be apprehended.

A retired medical officer of great Indian experience considers that, early in his career, he saw yellow fever in India. But I think it evident that his were cases of the disease which is now generally recognised as Indian relapsing fever, which stands widely apart from true yellow fever, especially in the particular that it is epidemic and without any definite gastric complication, whereas yellow fever is, with

¹ Surgeon Carleton has vividly described his sufferings under full quinism in enteric fever. *I. M. G.*, March 1886.

² 'Annual Report of H.M. 80th Regiment for the year ending March 1853,' by J. R. Taylor, Esq. *I. A.*, No. 2, p. 1.

some marked exceptions, under ordinary circumstances, endemic, and is characterised by black vomit. No one expects to meet with true yellow fever in India ; and its occurrence in that country has not been demonstrated by any medical authority.

DYSENTERY.

Our knowledge of the *causes* of dysentery is large, but inexact. The disease is very prevalent in the East and West Indies and in China. It occurs epidemically and endemically. In old times, it committed great ravages in the United Kingdom. I believe that the last general prevalence of dysentery in London was during the seven years 1840-47, when Dr. Baly examined post-mortem many hundred cases of the disease in the Millbank Penitentiary.¹ It is now said to have quitted London ; and, within the last three years, two distinguished hospital physicians of middle age have said to me, nearly in the same words, 'I have never seen a case of dysentery originating in London.' Still, at least while I used to consult them, up to about a twelvemonth ago, the weekly mortality returns used to report numerous cases. There is every reason to believe that dysentery may again ravage London at any time. Wherever it is or has been very prevalent on land, the influence of marsh poison may be traced. It is evidently a congener of paludal fever, and generally its worst expression in armies, in ships, and prisons, and in certain districts in India (as in Scinde, Behar, and Lower Bengal) and elsewhere, is largely due to scurvy. It has been attributed to brackish and putrid water, as cask-water, and to water contaminated by dead bodies ; but probably such waters, as well as fruits and other unwholesome food, act only as *exciting* causes. Ship and camp flux was mainly scorbutic (in some of these outbreaks the disease may have been enteric fever). Besiegers generally had flux, being exposed to the worst influences of climate and vicissitudes of weather, wanting proper food and water, and being kept 'stewing in their own juice' in the marshes which formed a main part of the defences of beleaguered places. In old military histories, we read little of flux among the defenders, who, until they actually starved, probably had a supply of vegetables from the gardens which, as scores of ancient plans which I have shown, appertained to almost every dwelling within their fortifications.

The Sanitary Commissioner's Report gives the following as the admissions and deaths, under the heading of Dysentery, in the two years 1881 and 1883.

¹ For data which I have collected regarding dysentery in London see papers contributed to the *Transactions of the Epidemiological Society* in 1882-3, 4 and 5.

	Admissions	Deaths
Year 1880.—European soldiers	2,865	177
„ women	111	3
„ children	135	13
Native soldiers	11,615	316
Prisoners	10,371	1,209
Year 1883.—European soldiers	1,495	21
„ women	69	5
„ children	109	12
Native soldiers	4,499	53
Prisoners	7,012	455

The whole of the known facts appear to favour the opinion that, at its first onset, dysentery is, in a constitution predisposed by marsh malaria or scorbutus, a simple catarrh of the mucous membrane of the large intestine, generally brought into action by chill arresting perspiration and causing portal congestion. The concurrent action of irritating ingesta and of long-retained feces is also frequently operative; but it is needless to say that, unless there be strong predisposition to altered function, the mucous membrane of the large intestine is exceedingly tolerant of irritation. The most curious thing about this catarrh, however, is that, of all catarrhs, it is the most easily curable if treated promptly. If dysentery be a specific disease, and a constitutional malady allied to fever, how comes it that, when taken within the first twelve hours, a single dose of ipecacuanha or of castor oil and opium very generally cures it, as if by a charm; yet still, if such appropriate treatment be neglected, even for a few more hours, the disease becomes one of exceeding severity, obstinately resisting treatment, and probably going on to serious follicular lesion?

Unhappily, we can expect to see cases of dysentery, at this its earliest stage, in exceedingly few cases, as in officers and their families and in our own persons. It is singular that we should not be more frequently consulted early in a disease which is very painful at its onset; but Europeans and natives generally undergo some course of popular treatment, which is, at best, ineffectual, before they come to us. The following is a common example:—At Chittagong, I one evening received a note from a young civilian asking me to send him medicine for a belly-ache. This gentleman's notes were usually written upon rough bits of paper, but this one was inclosed in a handsome envelope, sealed with a large coat-of-arms and unaddressed. From this, I inferred that Mr. S. considered the illness to be a serious one, and I went over to his house immediately. I found him walking to and fro his room evidently in great pain. His stools were dysenteric. In reply to my questions, he said that he had been ill all day, but had done his work in court. He had eaten nothing since morning but some *plantains*! To relieve the griping, he had taken *two glasses of brandy*! Knowing his careless habits, I felt certain that he would do ill unless I watched him carefully; so I took him at

once to my own house, where he got quite well slowly. Had he been attacked when out in the district, I doubtless should not have seen him for four or five days, during which he would, in all probability, have swallowed everything most likely to aggravate the dysentery, perhaps including some panaceas recommended by his servants. In my experience, patients usually applied for relief at about the fifth or sixth day, when nothing better than slow recovery can be expected. During the time thus lost, European soldiers often drink spirits freely.

When suffering from his fatal illness, Cardinal Wolsey, who was evidently well acquainted with the usual course of sloughing dysentery, said to Cavendish: 'I have a flux with a continued fever, the nature whereof is that, if there be no amendment within eight days, either excoriation of the entrails will ensue or frenzie, or else present death, and the best of them is death, and (as I suppose) this is the eighth day.' He died almost immediately afterwards. Dr. Edward Goodeve used the same expression to me. 'In a neglected case, sloughing generally occurs on the fifth or sixth day.'

Dr. E. Goodeve's Researches in Dysentery.—When I first took charge in the Medical College Hospital, I understood that my colleague, Dr. Edward Goodeve, had made very important observations on dysentery. These were never published. Writing to me, in September 1879, he said: 'I have written nothing on dysentery, to my sorrow. I have often intended to publish something, but procrastination has been my enemy.' I never heard a statement of his views. Hence it might appear that his research is buried with him, unless his students have kept full notes of his lectures, which I am not aware that any have done. Still, I think that it is in my power to give a fair idea of some of his principal conclusions. Thus my house physicians had been taught by him the method of *washing the stools*. I believe that, even now, many medical men in India have not adopted this most valuable aid in practice, and that it is still unknown in England. The stools passed since last visit have water poured freely upon them. Any feculent matter which may be present floats, the water is poured off, and whatever has sunk to the bottom of the vessel is allowed to remain there. This sediment contains the 'dysenteric products.' Water is again poured on and off until the 'products' are left nearly clean. These products are then placed in a large white dish filled with water, which floats them out and apart and displays them clearly. Here it is to be observed that, with the exception of feces and fluid blood, of which we, of course, take due notice, everything that is worth seeing and which tends to acquaint us with the nature of the case, sinks to the bottom of the vessel when the stool is washed. Holding a long pair of dissecting forceps in one hand and a long probe in the other, we examine the dysenteric products as they lie under water. These products consist mainly of

sloughs of various character and *mucus* in various conditions. My house physicians used Goodeve's nomenclature in describing these products. There were 'pus-infiltrated sloughs,' 'tubular sloughs,' and 'cellular or areolar tissue sloughs'; then there were 'rose mucus,' 'branny mucus,' and 'ropy mucus.' Besides these products, we generally found, with the washed sediment, any articles of food which had passed undigested, and which we always knew had greatly irritated the diseased bowel. Among these, the most common were coarse black pepper¹ from beef-tea, lumps of potato, raisins and currants from cake, cloves, &c.;—we often saw some of the ipecacuanha powder, or, when they were hard, the pills which we had given. We had thus the means of judging with fair precision whether the patient had obtained unwholesome and forbidden food surreptitiously. Too often, these patients lied unblushingly. 'Sir, I give you my honour as a man I have not tasted potato or cake for a month!' Then the washed matter would be shown to him quite unchanged, and he would either acknowledge that he now recollected that he had got one mouthful some days ago from another patient or would flatly deny that the stool was his. In that case, a student was placed to watch until he was next moved and immediately to wash the stool. This practice of washing the stools afforded us an almost perfect knowledge of the patient's condition. Having examined the products, we saw almost as much of the case as the surgeon sees when he looks at an ulcer on the cornea. Besides seeing what was happening within the patient's body, we could, without asking a question, perceive, almost to a day, the age of the case, and could prognosticate its final result with much confidence. Thus a quantity of gelatinous rose mucus with feculent matter—a case of not more than twelve hours' duration. The same mucus without feculent matter—probably this case has gone on unchecked by treatment for from six to thirty-six hours longer than the last. Branny mucus—this is a case from five to eight days' old, doing well under treatment. Ropy mucus in great abundance—a very favourable case approaching recovery. Pus-infiltrated sloughs—a case of five or more days' duration, in a tolerably good constitution, which will probably do well under treatment. A brown or black cellular slough, scarcely distinguishable at first sight from an old black cobweb pinched together to the same size and placed in the water beside it—an evidence of gangrene and presage of certain death. Now I cannot precisely say that these were Goodeve's observations, but I always regarded them as his. Having said 'wash the stools,' he gave the key and placed everyone in the right position to recognise the 'products' and to read their meaning.

¹ Among the quaint but sad facts of last century practice in India is this: 'Curtis ordered his dysenteric patients to eat pillaos, curries, grilled fowls, and peppered *chicken broth*' (H. H. Goodeve).

At p. 152, No. 18, of the 'Indian Annals of Medical Science,' my late colleague, Dr. Chuckerbutty, who had worked much with Dr. Goodeve, gave an account of the appearances revealed by washing dysenteric stools. It was long considered that this paper contains Dr. Goodeve's observations. The author, however, does not state that it does; and, in the letter which I have cited above, Dr. Goodeve wrote: 'Chuckerbutty published a paper, but, beyond saying that I had washed the evacuations, did not add much to our knowledge. . . .' 'He drew no inferences that would be of much use.' I think that, in the face of this criticism, we must not say that Dr. Chuckerbutty's paper represents Dr. Goodeve's views. Still this monograph, as coming from a careful observer of the Goodeve school, deserves the close study of all who desire to work out the morbid anatomy of dysentery. When it was first published, one or two of our friends told me that they thought the author's description of the products was rather fanciful. I replied that, although I did not read all the products precisely as he did, I had repeatedly seen, and was perfectly well acquainted with, nearly every product which he described.

I think that the *symptoms, morbid products found in the dejecta, conditions of the intestine, prognosis and treatment*, may best be considered if we describe the disease in stages.

Catarrhal Stage.

We generally find that, although usually in rather poor health, and probably, in India, liable to attacks of intermittent fever, the patient has done his work and taken his food nearly as usual almost up to the moment at which he is attacked. There may be chilliness, but I have never seen or heard of rigor. Usually late in the evening or after going to bed, there is one very copious semi-solid evacuation passed without tenesmus or griping pain. The large intestine has emptied itself completely or nearly so by a strong peristaltic action. After this, the patient may fall asleep, but is soon awakened by sharp tormina, and passes, with great and painful tenesmus, an ounce or two of 'rose gelatinous mucus,' with or without feculent matter. During the next few hours, there are frequent discharges of this mucus, attended with ever-increasing griping, straining, scalding, and bearing-down action. In certain cases, the mucous stools are passed with very little or no tenesmic suffering. I have seen a child running about at play, and now and then passing a heap of mucus on the mat. I usually found that these cases did more or less badly. I was in the habit of saying, the greater the tenesmus and pain, the higher is the vitality and the more favourable the prognosis.

Citing an interesting passage from the records of the Madras Medical Board in 1807, Mr. Cornish observes: 'We see, now-a-days, occasionally, dysentery of the true hæmorrhagic type; but it is not a

common thing to find, as did Mr. Heward, in H.M.'s 30th Regiment at Wallajabad, that men 'on guard, at parade, or in bed, became first cognisant of the existence of their malady by passing a large quantity of fluid blood unattended with griping or tenesmus.'

In some cases, at this time, there was the condition very usual in Indian dysentery—tenesmus and tormina after the first or second evacuation with febrile symptoms; but Mr. Heward said that 'most frequently the disease comes on in the shape of common diarrhœa. The man, while the complaint continues so, apprehending nothing from it, seldom reports himself till he sees an appearance of blood, or blood and slime, mixed with his stools, and there is, in this stage, commonly a dull heavy weight, rather than pain, over the hypogastric region, with sometimes an occasional gripe; but, when this happens, which is by no means constant, on a more plentiful discharge of blood, the griping very often entirely ceases, leaving now and then tenesmus, which continues more or less troublesome throughout the course of the disease.'

I apprehend that the occurrence of almost painless dysentery has led to the, as I believe, absolutely erroneous idea which I have heard mooted by men of authority in London—never by any Indian medical man—that there exists a destructive malady which is designated as '*dysenteric diarrhœa*.' If there be such a disease as this, I have still to become acquainted with it. I know diarrhœa and dysentery only as perfectly distinct entities, always to be differentiated, if not by watching the symptoms, certainly by washing the stools.

At the early stage of typical Indian dysentery, there is generally a sense of sickness. My own sensation was that the pain nauseated me. The pain of colic has made me retch, but not dysenteric pain.

The rose mucus very much resembles thick arrowroot, more or less tinged and spotted with blood. The probability is that the inflammatory action usually commences first in the rectum and sigmoid flexure and rapidly extends upwards. In a large experience, I have not had the opportunity of examining the intestine at this, the very earliest, stage of dysentery; but my friend, Dr. Joseph Ewart, of Brighton, has traced 'the gelatinoid exudation, termed by others "gelatinous mucus," free from or tinged with blood,' as coming from enlarged solitary glands.

All the best authorities of the present day agree in considering that dysentery is, in the main, a morbid condition of the follicles. At first, this must be only functional, as a single dose of medicine often cures it. Dr. Bleeker observed hyperæmia of the mucous membrane by means of a speculum ani. Dr. Bleeker's memoir on dysentery appeared, in its English form, in 1854, in the first number of the '*Indian Annals of Medical Science*.' It was long regarded, in India, as our best description of the malady. I, however, think that its author

erred in not showing that the lesions commence at the follicles and centre there. The paper may, however, be studied with great advantage, as being the work of a scientific observer who, personally, made post-mortem examinations in many cases. A diphtheritic exudation, coating the mucous surface of the bowel, as an exudation tube covers the mucous membrane in croup, is described by Bleeker and Virchow. The former had not observed such cases until after he first published his memoir. I long sought in vain for anything which I could regard as 'fragments of croupous tubes (and sometimes large ones),' which led the patients 'to imagine that they had passed pieces of the intestine.'

I attach great value to Dr. Ewart's description¹ of the state of the intestine in dysentery, and shall follow it in these notes. Ewart teaches that 'the *first* visible change in the solitary glands is congestion, the vessels surrounding and penetrating the capsules being turgid and engorged with blood. The *second* change is augmentation of their contents from the accumulation of albuminous exudation and enlargement from the size of a millet seed to a small shot (Baly). The *third* stage is . . . rupture of some of the capillaries in the interior of these little vascular glands, extravasation of blood, with the area of the ordinary dark point on the free aspect increased.' 'In a large number of instances, the morbid process may stop short, under proper treatment, at any of the first three stages, and repair is then effected by resolution.'

As I have already said, the majority of cases yield to treatment if seen at this stage of the disease. In opium and ipecacuanha, of which the latter is the most valid, we possess two powerful antidysenterics. A brief analysis of the history and uses of these antidysenterics will occupy a brief chapter.

Having repeatedly experienced its curative power in my own person, I always gave, at the first onset of dysentery, a draught, recommended by Dr. Scott, and prescribed for myself by the late Dr. Henry Marshall Hughes, composed of 25 minims of laudanum, and two drachms each of castor oil, and compound tincture of rhubarb in cinnamon water. Supposing this to be taken in the morning after a night of suffering, the patient, remaining warm in bed, usually has some hours' sleep. Immediately the bowels act once, probably about evening, the evacuation being usually without griping or tenesmus. Three grains of opium, rubbed up in an ounce of thin starch or mucilage, are now administered as an enema, care being taken that the tube enters beyond the sphincter. This must be retained. Next morning, the patient usually wakes cured; but it is needful that he should, for three or four days, confine himself strictly to a recumbent position—

¹ Quain's *Dict. of Med.*, Art. 'Dysentery.'

a most important precaution in every stage of dysentery—and be restricted to a very moderate milk diet. An extra tablespoonful of tapioca pudding gave me a severe relapse. Barley water, made with Robinson's patent barley and sipped, is the best drink. For feverishness, Dr. Goodeve gave drachm doses of solution of acetate of ammonia.

Ulcerative Sloughing Stage (as distinguished from gangrene of the colon).

When I first saw a patient, at about the fourth day of his attack, I found him very ill, and probably began a course of ipecacuanha. In a day or two he would look cheerful and tell me that he felt much better. On washing the stools, I would find masses which very closely resembled in size, shape, and colour, rotten oysters; there might be one or more of these with fragments of the same character. Lifted with forceps, these fell back into the fluid with a 'flop.' The largest of these would be about the third of an inch thick, their edge was perfectly defined, and they consisted of the whole depth of the mucous membrane with the submucous connective tissue and perhaps some striae of the muscular coat. The name of 'pus-infiltrated sloughs,' which Goodeve gave them, precisely indicates their character. In other cases, enormous *tubular* or plane sloughs would pass. Not infrequently, these great sheets of mucous membrane, many inches long, remain hanging from the anus for hours until, after we have cut them off, the attached part separates of itself or is drawn away cautiously with forceps. It often seemed to me that (under ipecacuanha) these patients' general condition appeared to improve in proportion as they passed more and more of these forms of slough. It is often astonishing, even to an experienced physician, what immense quantities of the mucous membrane, which the tubular slough represents, can be passed while the patient's general state is clearly advancing to recovery. I had in my ward a strumous-looking druggist's shop-man, not long arrived from England, who, in a few days, passed so much mucous membrane that it really seemed almost incredible that the large intestine had it to throw off. Several sheets of massive tubes and planes passed with the greatest ease. His recovery was not remarkably tardy. He came to me again about a year later, fat and healthy-looking, but suffering from great rectal contraction. On going out, somewhat relieved, he married one of the nurses. The loss of mucous membrane is often so terrible that these cases can hardly do well eventually. The ulcer from which an oyster-like pus-infiltrated slough has separated may leave a beautifully even cicatrix which may almost disappear in course of years, but I have seen cases in which the loss of mucous membrane and the subsequent contraction were so great that the enterotomy scissors with great difficulty found the course of the gut, which, when it was at last

laid open, had an appearance which I can only compare to that of a stocking, large holes in which have been—not darned—but dragged together with a needle and thread. ‘These constrictions,’ says Ewart, ‘are frequently the mechanical cause of constipation and faecal accumulation.’ In many cases, the sloughing and ulcerated bowel becomes enormously thickened. A native, having passed dysenteric stools, clearly became the subject of intestinal obstruction. I found a massive, doughy, sensitive tumour extending throughout the ascending colon into the arch. In shape and size, it resembled a thick and large Bologna sausage. Just previously, I had in my ward two native men each of whom died from invagination of a wandering cæcum into the arch of the colon. This man’s condition, outwardly, so much resembled theirs, the swelling having precisely the same proportions, that I thought I had a third case of intussusception. The autopsy revealed dysenteric disease of the ascending colon, which was attended with such massive infiltration of the tissues as to render the bowel practically impervious, although its greatly narrowed canal admitted the scissors. This case of obstructive infiltration is unique in my experience.

When the sloughs have separated and the ulcers which they leave are granulating and cicatrising, the ‘products’ will be seen to contain, in more or less abundance, particles which have precisely the appearance of bran steeped in water, the ‘branny mucus’ of Goodeve. This was always regarded as a favourable sign. More rarely, the pot is found to contain a large quantity, a pound or two, of what Goodeve designated as ‘ropy mucus.’ This has the appearance of pure white transparent fluid gelatine; its tenacity is such that the students used to show it by pouring some from one pot to another and then separating the utensils, the margins of which would remain connected by a bridge of mucus, self-supporting midway and several inches long. When this appeared, speedy recovery was always expected. Such mucus is not infrequently secreted in nasal catarrh, just before recovery.

Ewart shows that the *fourth* stage of follicular disease is marked by atrophy and molecular disintegration of the free aspect of the capsular wall and escape of the morbid gelatinoid blood-tinged contents into the canal of the intestine. This is the rule, but, in very exceptional cases, the capsule may burst through the attached portion, lighting up inflammation in the neighbouring connective tissue and muscular coat. ‘In many cases the morbid action is cut short after the completion of the fourth stage, without further extension of the disease. The adjoining follicles of Lieberkühn do not, in these cases, necessarily participate, to any great extent, in the diseased process. Under these conditions, when the whole of the exudation has been expelled, the glands regain their tone and functions, and recovery, rapid and complete, ensues.’

It was stated, some years ago, by a French observer, that dysentery

does not occur twice in the same subject. This is altogether erroneous. I have had five attacks—always promptly checked. From experience in my own person and from the observation of many other cases, I feel certain that, where the disease is cut short previous to the sloughing stage, recovery from dysentery is as complete as from nasal catarrh.

Ewart goes on to show, that, when the disintegrating or ulcerative process is developed, 'the whole of the solitary glands engaged perish. The ulceration involves the neighbouring tubular glands, leading to ulcers varying from the size of a mustard-seed to that of a florin or more; in depth, generally extending to the sub-mucous connective tissue and not infrequently laying bare the circular lamina of the muscular coat, sometimes involving the longitudinal layer and perforating it as well as the peritoneal coat.' I have already described the large tubular sloughs.

Subacute Stage.

I never could convince myself that there is such a disease as '*Chronic*' *Dysentery*. Given a colon with ulcers indisposed to granulate and cicatrise, the evacuations are generally lax, but not dysenteric. From time to time, at intervals which generally become shorter as the malady proceeds to an unfavourable termination, there is a distinct attack of acute dysentery. I used to tell my students that a row of beads, irregularly strung on a single thread, represented, diagrammatically, the sequence of the acute attacks in a case of subacute, or so-called chronic dysentery. Having a case of this kind under close observation in hospital, I could generally command each acute attack as it occurred, commencing with a dose of castor oil and laudanum, which swept down the undigested food which was nearly invariably present as an irritant; and, regulating the diet, I often succeeded in checking the attack without ipecacuanha. It appeared to me that, at this point of the disease, we had to deal with commencing dysenteric ulceration in that portion of the mucous membrane of the colon which had hitherto escaped ulceration and with an irritated state of the ulcers which had remained unhealed. I have never—probably because I treated each attack promptly—seen here the pus-infiltrated oyster-like sloughs of ulcerative action; but, occasionally, a case of this kind, brought in late, ended by extensive gangrene and collapse. I believe that, if these patients would only remain under treatment, and *take none but proper food*, they would probably nearly all recover, whereas they nearly all do badly. With an intense and apparently uncontrollable craving for the most unwholesome food upon which they can possibly lay their hands, and with digestive power so impaired that even the best chosen and blandest food will scarcely agree, native patients will swallow, almost unchewed, handfuls of horse-beans (*gram*), and Europeans will, as I have said above, bolt cloves, raisins,

currants, and large masses of potato. A lady, at about the fifth month of pregnancy, was in this subacute stage after an acute attack. As she lived at a considerable distance, I brought her into our own house that I might watch her progress steadily. Despite treatment, and, as I thought, well-regulated diet, the disease advanced steadily. She aborted and died. It seemed to me that my hands were absolutely tied in this case. The difficulty was afterwards explained when I discovered that her ayah had systematically supplied her with the most indigestible meat and fruit, such as roast pigeon and pineapple, swallowed in crude masses. Had I then known Goodeve's practice of washing the stools, I should have been able to check this deadly voracity.

Dysentery of tubercular origin is the usual complication of pulmonary consumption in India. When, as frequently occurs, the rectum is closely beset with irritable ulcers, the patient's suffering is extreme. Some alleviation may be obtained by the application externally of opiated oil and of opium cataplasms, and by the use of opium (not laudanum, which causes irritation) enemata.

Colitis—gangrenous dysentery.—In common with several of my Indian predecessors and contemporaries, I have always been uncertain whether this terrible disease is the worst expression of dysentery in broken (usually scorbutic) constitutions or an independent malady. Having repeatedly seen it follow an ordinary attack of dysentery, I am strongly inclined to believe that it is the gravest form of that disease. Thus a dear brother officer of my own was attacked with dysentery in the rainy season. There was a marked tendency to phthisis in his family, but he enjoyed good health until just before his fatal attack, at about the age of forty. His practice increasing rapidly, he was overworked, and I thought lived poorly, drinking fruit syrups in water or soda water far too abundantly. He recovered with some difficulty, resuming his work and going into society; but he was pale and languid, and always had *two formed stools* in the twenty-four hours. When this occurs in Europeans (it is natural in rice-feeding natives), a return of dysentery, upon a chill or indiscretion in diet, is always to be feared and guarded against. October set in wet and stormy, and he was attacked with gangrenous colitis, which proved fatal in nine days. In these cases, enormous gangrenous sloughs are either passed after about the fifth day, or are found *in situ* after death, the colon lying collapsed and mortified in all its tissues, with marked traces of colic peritonitis, like a dead and putrid snake. Often, in these cases, the black, flocculent, cobweb-like slough of areolar tissue is passed, sometimes alone, at others attached to a mass of dead mucous membrane. I repeat that I always regarded this as a certain presage of death. With the sloughs there is a large discharge of cruor, having the consistence and appearance of a mixture of cream and blood, and a horribly and

very characteristic gangrenous odour, which, after one experience, can never be mistaken. Collapse, and the hippocratic visage, which may last from twelve hours to three days, precede death. Nearly all these cases prove fatal within about ten days.

Treatment.

Isolation.—Unquestionably, cases of dysentery ought not to be herded together, and ought, for their own sake and for that of others, to be kept apart from the subjects of other diseases. A great deal was said by old writers about contagion in dysentery. Harty¹ appears to have agreed with those who regarded the dysentery of London which followed the Great Plague as contagious and malignant. Dr. John Clark, who published the results of his experience of Indian disease in 1792, and afterwards practised at Newcastle, mentions that, in the year 1781, dysentery was introduced into a dockyard in that neighbourhood by some sailors who returned from abroad ill of the complaint. The disease soon spread amongst the workmen, and several died. Aitken cites Maclean to the effect that, in the old infantry barracks at Secunderabad, long notorious for a severe form of dysentery, it was observed that men labouring under other diseases who happened to be exposed to the putrid effluvia of the excretions of dysenteric patients were often severely affected by the disease. I never saw any positive evidence of contagiousness in Indian dysentery, but I attach great importance to the precaution, insisted upon and strictly practised by my late friend Dr. John Sutherland, of not massing dysenteric patients together, and of strictly segregating every case of gangrenous colitis in a separate building. I believe with him that a patient with ordinary dysentery, admitted to a ward foetid with dysenteric dejecta, is almost certain to fare ill; and that such a case, placed in the atmosphere which surrounds a patient with gangrenous colitis, can scarcely escape death. When I inspected the large jail at Patna, I found Dr. Sutherland's hopeless cases of dysentery well cared for in an out-building. Although we could never convince Government of the fact, nothing can well be more irrational than the practice which obtained in the Medical College Hospital up to the time at which I left India, of placing surgical patients in an atmosphere charged with dysenteric exhalations. I put up valved doors and a party wall between my ward and the surgical wards:—but, when I saw, upon the direction of a patient's letter, '*Dr. Chevers's Dysentery Ward*' and, looking around me, knew that the ward was, at that and many other times, correctly designated, I could not but feel that my patients and I were out of place.

Mr. Scriven holds that, when dysentery has gone on to ulceration, 'ipecacuanha, unless it be in one or two grain doses, is no longer useful.' This, like all others of my friend's opinions, commands great

¹ *Observations on Simple Dysentery*, p. 93.

deference, but it does not accord with my own experience. Nearly all my hospital cases came to me late with ulceration, and I gave ipecacuanha freely as long as the slightest trace of dysenteric mucus could be discovered in the stools, save always when the case proved to be gangrenous colitis, in which I knew ipecacuanha to be useless. Indeed, had I not done this, I should only have found opportunity for using the drug in private practice. Most of those thus treated did well sooner or later. In this stage, Mr. Scriven relies upon large enemata, with alum, and the bael fruit. He also employs opium by the mouth, and in small enemata. The treatment of dysentery by Ipecacuanha is specially treated in another section, page 159.

Diet.—My father, who had large experience of dysentery in the West Indies, said that soup is poisonous in this disease. In all forms of dysentery, *salt* is likely to act as an irritant.¹ In supporting patients for weeks by the use of strong beef-tea and egg enemata, I found that, whenever salt was used, the bowels became loose, pepper and other spices act simply as irritants and may always be detected in washing dysenteric stools. These facts appear to me to explain my father's objection, seeing that strong unseasoned jugged fowl, soup, taken in good quantity but little at a time and cold, always appeared to agree well with my patients in the sloughing stage immediately ipecacuanha was tolerated. In the earliest stages of the disease, I gave little but milk diet and patent barley powder. The old doctrine, that all drugs and articles of food containing matter likely to reach the abraded colon in an irritating solid form is hurtful, cannot be too attentively observed.

Twining commented upon '*the cruelty of embarking persons in a hopeless state, who are susceptible of no change except aggravated misery by the removal.*' Although the most friendless sick in India are generally cared and provided for with brotherly kindness, I have, in my voyages to and fro, seen only too much of the anguish unnecessarily endured by patients injudiciously sent to sea in the vain hope that they may recover from a moribund condition which ought to have been self-evident—in empyema, hepatic abscess, advanced dysentery. In a case of the last kind, the sufferer did not survive embarkation twenty-four hours, and the step lost to his estate some hundred pounds which it could ill spare. At least until I left India, there was no special accommodation in the best passenger vessels, as there ought to have been, for dying passengers—although the officers spared no effort to do what

¹ The generally recognised fact, alluded to in the chapter on Cholera, that all new arrivals in Calcutta and Delhi used to be liable to attacks of bowel-complaint depended upon the saline impurity of the waters of those places. At one time persons leaving India in the P. & O. steamers were liable to dysentery, attributed to drinking condensed sea-water. The founders of one of our great Australian cities—I think Melbourne—suffered much from dysentery caused by drinking brackish water.

was needful. Science and humanity alike teach that, when the condition of a patient is self-evidently hopeless, he ought to be allowed to die quietly in his own bed. Still change often effects marvellous cures—in no cases more signally than in dysenteric patients who are not moribund.

Twining makes the following excellent remarks upon the good effect of a sea voyage upon old cases of dysentery: 'A voyage to sea often restores them when nothing else will. A similar remark has been made with respect to some descriptions of protracted dysentery in the West Indies, which commence recovering from the moment they sail. Desgenettes gives a very remarkable instance of the same sort in Egypt, when 400 cases of chronic dysentery were sent on board ship from Alexandria. Nineteen of these patients died within a few days of embarkation, the rest all recovered. Have we any means of ascertaining in what stage of dysentery patients are unlikely to receive benefit from going to sea?' Children and the subjects of ordinary sub-acute dysentery frequently recover either on the voyage home or soon after landing. I have frequently watched the rapid improvement of utterly emaciated little children on the overland passage; and the long sea voyage, on a well-found ship with a good medical man, is still better. The otherwise uneasy motion of a screw-steamer, sailing-ship, carriage or palanquin, generally tends to cause constipation in healthy persons, and usually assists recovery in dysentery. Embarking at Marseilles for India, in the winter of 1867, one of my fellow-passengers was a very sickly-looking elderly gentleman, an indigo-planter long resident in the East. He told me that, during many months' stay in England, he had been constantly tormented by 'diarrhœa' (I did not see the stools). Outside Marseilles, we had very stormy weather, and the steamer was in danger. Nevertheless Mr.—— steadily improved. His bowel complaint left him and remained absent (he consulted me for another disorder many months after reaching India) and, before we landed, he had become one of the most active and cheerful members of our society. A barrister told me that he was suffering much from dysentery at Allahabad, when he found, to his horror, that he must immediately travel, by palanquin, to Calcutta—a most tedious and fatiguing journey of several hundred miles. He fully expected that he would die on the way. He took a dose of opium at starting, and he was astonished at finding that he grew better as he travelled. He was well when he reached his destination. So much was passive motion valued formerly by Indian physicians, as a means of 'shaking down the liver'—doubtless on the principle which led one to say that his chief doctor was a horse, and his second an ass—that the verandah of nearly every wealthy house had its long spring board on trestles. It was often amusing to see a portly judge moving airily up and down in this exercise.

Sydenham fully recognised the most important principle that the recumbent position is absolutely needful in all stages of dysentery. In every case of this disease, the condition of the liver should be daily examined, that any threatening of congestion, tending to hepatic abscess, may be recognised immediately and be promptly dealt with. We now and then meet with sub-acute cases of dysentery, where there is little or no dysenteric ulceration below O'Beirne's sphincter which separates the sigmoid flexure from the rectum. Here there may not be frequent stools. A solid feculent mass as large as a goose's egg will form in the rectum and be passed from time to time, having upon it a heap of dysenteric mucus, which follows it from the almost strictured sigmoid flexure. I have found a few ounces of very strong black tea, used as an injection, efficacious in these cases.

The congestion and irritation of pelvic organs which dysentery maintains, especially where the rectum is the seat of extensive ulceration, frequently produce serious consequences. Dysuria is often complained of in rectal dysentery. A very accomplished medical man, who died of dysentery shortly after his arrival at Calcutta, suffered from such extreme distress at the neck of the bladder, that he declared himself to be the subject of stone. Before large doses of ipecacuanha came into use, a young lady, born in India of European parents, and apparently of low constitutional power, began to menstruate and to suffer from hæmorrhagic dysentery at the same time. The two discharges went on in great profusion, and she died in about ten days. Young married ladies, arriving in India pregnant, are frequently attacked with dysentery. When morning-sickness is present, and abortion appears to be imminently threatened; or when, later in pregnancy, the death of the fœtus and premature delivery have to be guarded against, these cases are fraught with difficulty and anxiety. The morbid capriciousness of these patients' appetites, and the perversity with which they crave for unwholesome food add much to the physician's embarrassment. Our best hope is to see these cases early, and to arrest the disease by castor oil and laudanum and by opium enemata. Ewart's experience of these cases satisfies him that large doses of ipecacuanha are indicated when dysentery occurs in pregnant women (Quain, p. 414). Here, as in other cases of dysentery, he objects to treatment by opium, save by opium enemata to relieve local irritation. Still, in my own practice, morning-sickness was always a formidable bar to ipecacuanha treatment; later in pregnancy, the prompt and steady use of ipecacuanha may be hopefully employed. A valuable paper on 'The Management of Tropical Dysentery and Fever in the Latter Months of Pregnancy' was published in 1839 by Dr. Duncan Stewart,¹ who showed that dysentery is 'not unfrequently overlooked by the patient and neglected too long, being mistaken for

¹ *I. M. J.* 1839, p. 587.

some of the common bowel derangements to which women are subject at such times.' I lately heard of a lamentable case in which a young lady, recently arrived in India, suffering greatly from morning-sickness, did not seek aid for four days, when the dysentery had become hopeless. She sank on the seventh day. Modern practitioners will hardly agree to adopt Dr. Stewart's chief means of treatment. He wrote, 'The lancet is, of course, our sheet-anchor'; and, 'Relief by bleeding is almost universally afforded in the urgent complaints of pregnant women, even in constitutions which, at other times, do not well bear that evacuation'; and again, 'Venesection should be practised always in the recumbent posture, to avoid syncope.' He gave a full dose of calomel after bleeding, followed by blue pill. We can more readily value his advocacy of opium enemata, rest in the horizontal position, quiet, and warm fomentations. He adds, 'If acute dysentery be not thus early arrested, and is allowed to persist for some days, it will certainly produce abortion, and this often very suddenly and unexpectedly; the first stage of labour, *i.e.* the expansion of the os uteri, having probably been going on unperceived for several days. I have known labour to be completed in such circumstances in less than five minutes, with not more than two regular pains. The labour will generally come on just when the dysentery is itself about to yield to the treatment, and we have begun to flatter ourselves that all is now doing well. The danger of immediate death is imminent from prostration and loss of blood. The importance, therefore, of an early and active treatment of dysentery is apparent, and of perpetual watchfulness until the cure is fully established.'

I do not think that much need be said, in the present day, in reprobation of the old practice of *blood-letting* in dysentery. I never saw it employed. The experience of my own repeated attacks convinced me that no course of treatment could well have been less appropriate in the types of this disease which I have seen. Morehead, however, recommended venesection with great caution, and said that 'in cases in which, from the state of the system, general blood-letting is contra-indicated at the commencement of the attack, recourse may be had to the application of leeches, in numbers from two to six dozen' (of the very small Bombay leeches), 'repeated more or less frequently according to circumstances' (page 294).

I will not question that types of dysentery may have prevailed in which venesection and leeching were really indicated; but most assuredly they were not indicated or to be justified in such cases as the following, which is taken from an old record:—'1st January. Ill three days. *Fiat v.s. ad deliq.* Evening, blister to the hypogastrium. 2nd, eighteen ounces of blood drawn, causing syncope. Pulse 100, feeble; xv leeches to anus. 3rd,—of opinion that the colon is, and was on admission, in a disorganised (almost gangrenous) state; a most

unfavourable prognosis. One measure of port wine, one pint of milk with sago. Spoon diet. *Vesp.*, died. The colon, from cæcum to anus, gangrenous.'

Treatment of Subacute Dysentery.

In the intervals (*vide* p. 149), quinine and astringents such as the permittate of iron, much valued by Dr. Maclean, and the bael (fruit of the *Ægle Marmelos*) strongly recommended by Sir Joseph Fayrer and Mr. Scriven, are likely to be useful in giving the patient strength and restoring the lost tone of the diseased intestine. I do not think that the use in dysentery of the raw pulp of the ripe bael fruit has found many advocates during the last twenty-five years; but, when I first went to India, this '*pucca*' bael was much used. My predecessor, when in charge of a jail hospital to which I succeeded, tried it fully. I was set against it, finding in the book several records like this: 'Peeroo—admitted with dysentery of six days' standing, one bael fruit. Next day, worse: Rep. bael,' and so, in like manner, for three or four days; and then 'Died.' My friend died of dysentery under my care and in my presence, but never proposed to take ripe bael. In native practice, the fruit has reputation as an antidysenteric, and we considered that, being one of the Aurantiaceæ, it might be antiscorbutic. I always valued a spiritous fluid extract of the green (*cutcha*) bael fruit dried upon a formula prepared by my friend Roy Kanye Lall Dey Bahadur. It is a bland aromatic astringent. By such means as those laid down at p. 149, a medical man, with strength of mind sufficient to resist the promptings of dysenteric bulimia, and with opportunity of returning to Europe, would, I believe, generally be able to free himself from subacute dysentery; but a very large proportion of adults eventually succumb to the malady. Probably because they are, for the most part, carefully watched and strictly controlled, and are sent to England early, a large proportion of European children bear subacute dysentery long and eventually recover.

Treatment of Gangrenous Colitis.

As this disease begins as common dysentery, we commence by treating it as such; but nothing that I saw done or did myself was of any avail. Ipecacuanha is quite useless. Opium is generally given, and upon a sound therapeutic indication, but it is not curative. It rapidly allays pain, but the disease is not always very painful. Believing scorbutus to be the leading causative agent here, I, many years ago, determined that all my patients with gangrenous colitis should have lime-juice freely, both by the mouth and by enema. It singularly happened that I gave the juice first to a sailor who was passing a great deal of blood, but who had no symptoms of gangrene. Hæmorrhage ceased at once, and he got well so rapidly that I believed that I possessed an

invaluable specific. But, with this solitary case, my success ended, although—considering that the *tendency* of lime-juice was to do good in these cases, that it never did the slightest harm, and that lemonade from the fresh lime was grateful to the sufferers—I gave it in every case until I left India.

*The Employment of Opium, Calomel, and Ipecacuanha in
Dysentery.*

The potency of *Opium* as an antidysenteric is unquestionable. Commensurate with the sanative efficacy of this drug is the extraordinary degree to which it is tolerated in dysentery. About the year 1796, C. Maclean gave it, in combination with calomel, in enormous doses. He is careful to mention that ‘the laudanum was weaker, perhaps one-third, than what is commonly used in Europe.’ In the case of a young man with dysentery of a fortnight’s standing and a threatening of liver abscess, he commenced with moderate mercurial treatment and ‘draughts of 70 or 80 drops of tincture of opium, repeated according to circumstances through the night. In the course of a few days, the calomel was increased to six grains, with two grains of opium, every two hours; an ounce of mercurial ointment was rubbed in at four times in the course of the day; and draughts with two hundred drops of tincture of opium in each were given every second hour during the night’—say forty-eight grains of opium in the twenty-four hours; and yet, our author says, ‘Some cases of dysentery will require much more than this quantity.’ ‘For a fortnight he seemed to get better;’ but the symptoms of liver abscess, which was found after death, increased. ‘The medicines, however, were continued.’ He died on the twenty-fourth day of treatment. Then we have a still more remarkable case, treated by Dr. William Yates, of the Madras Establishment, in 1796, in the Calcutta General Hospital. On August 21 the treatment of a man with dysentery commenced with ‘eighty drops of tincture of opium morning and evening.’ The opium was steadily and rapidly increased up to September 1, when ‘he was so extremely weak that Dr. Yates considered him as approaching to dissolution.’ ‘Draughts of five hundred drops of tincture of opium each were ordered to be given every hour,’ ‘with glysters of five hundred drops, to be repeated, according to his own discretion, as the tenesmus and griping might indicate.’ On the following day, ‘he had not slept, but felt himself better.’ The report is carried up to the 30th. ‘Being a sensible and steady man, he was, at that time, allowed to proportion the strength and frequency of the draughts and glysters according to his own judgment.’ Up to this time, at least, ‘he had an allowance of wine, from one to two or three bottles in the twenty-four hours, according to exigency.’ He is stated to have recovered, but it is not mentioned when. Another patient (case xxii.) recovered from

dysentery in a month and four days, after having undergone an equally extraordinary course of opium, wine, and mercury, the *rationale* of this plan of treatment being to effect a cure by employing that 'high degree of stimulant power which may sometimes be required to effect a cure in the state of exhaustion which constitutes dysentery.'

It will be noticed in the next section, on the use of ipecacuanha in dysentery, that, in 1807, Mr. Abercrombie used very large doses of that drug in combination with doses of opium equivalent to fifteen or eighteen grains. Dr. Cornish, who has brought this important record to light, explains that he has ascertained that the opium in use then was bazaar opium, grown in the province of Mysore, or the Hyderabad country. The Mysore opium was certainly of good quality, as it is to this day. He is doubtful as to the purity of the Hyderabad opium; but, late in the year 1807, in which they had considered Mr. Abercrombie's practice, the Medical Board arranged to secure regular supplies of 'Patna opium' for hospital use.¹

If Maclean and his contemporaries failed to prove the antidysenteric efficacy of large doses of opium, they, at least, demonstrated the fact that the system tolerates it marvellously in that disease.

Twining strongly condemned the use of opium in dysentery. Sir Robert Christison, who has only lately passed from among us, wrote: 'In the severe form which dysentery occasionally puts on in this country I have known a patient take from twenty-four to thirty grains of opium daily, and retain it all without experiencing more than a mild narcotic action.'

Kenneth Mackinnon was one of the last who advocated the treatment by opium alone. He evidently agreed with Maclean in attaching value to opium as a stimulant. He remarks: 'The constitutional effect of opium has often appeared to me in bad cases to counteract the depressing effect of the disease on the circulation. This applies chiefly of course to those violent cases where deep ulceration and sloughing cause death at a comparatively early stage of disease, and is especially true with regard to cases where the profusion of the discharges, bloody or watery, is acting injuriously.' He then recommends the use of acetate of lead, which is not, I think, employed in the present day, and continues: 'Where the functions of the brain are not impaired, but death is threatened by the extinction of the heart's action, I believe that opium is the sheet-anchor of treatment, preferable to stimulants probably by reason of its combining a sedative with the stimulating quality.' In 'sthenic' cases of dysentery Mackinnon began his treatment by 'free depletion.' He combined opium with mercury. 'For

¹ It appears in the Records of the Bengal Medical Department that, in 1790, Government directed that opium manufactured in Bengal, Behar, and Benares should, on arrival in Calcutta, be examined by a member of the Board, and reported on in concert with the superintendent of opium manufactures.

the procurement of sleep, the soothing of pain, and the temporary check of purging, one large dose '—three grains—[of opium] ' may be given at night, combined with blue pill or calomel if that be thought desirable.' Valuable as everything that he wrote is, this chapter of Mackinnon's especially deserves careful study. If I could not procure ipecacuanha, I should treat my cases of dysentery very hopefully by the *very* cautious use of opium combined with diaphoretics, one of which (nitric ether) I have used with great advantage in this disease. My friend Mr. Scriven successfully treated a case of dysentery, in which I was much interested, by large doses of morphia. I never ceased to value opium as an anti-dysenteric in combination with ipecacuanha. I used it most in three-grain enemata, and in the struggle which frequently meets the attempt to persuade the stomach to tolerate ipecacuanha. But latterly, although I never gave opium up, I gave it less, in the conviction that ipecacuanha is all-potent in dysentery.

Calomel in large doses has undoubtedly considerable power as an antidysenteric. Certainly, in doses of from ten grains to a scruple, this drug acts as a sedative upon the alimentary canal. Such reports as the following, by Mungo Park,¹ could not fail to recommend its use :— ' Ever since my arrival at Marraboo (on the Niger) I have been subject to attacks of the dysentery ; and, as I found that my strength was failing very fast, I resolved to charge myself with mercury. I accordingly took calomel till it affected my mouth to such a degree that I could not speak or sleep for six days. The salivation put an immediate stop to the dysentery, which had proved fatal to so many of the soldiers.'

Early in my career, I gave calomel in large doses (up to ten grains) in dysentery and cholera, I think with benefit ; at all events, the patients had good recoveries. Doubtless fashion had much to do in the maintenance of this practice of placing slavish reliance, in nearly all tropical diseases, upon ' the Samson of the materia medica,' as Dr. Chisholm designated mercury.² Still it is to its credit that the men of Twining's and Mackinnon's schools valued it ; but the fact, fully set forth in Halliday's Memorial, that patients with dysentery often died after weeks of suffering devoted to the misery of swallowing nearly a thousand grains of calomel, shows that, whatever its value may have been in judicious hands, it fell under the objection which applies to ' too much of a good thing.'

Ipecacuanha.—A glance at page 526, vol. ii., of that noble work of learning and research, ' Waring's Bibliotheca Therapeutica,' will show how universal the antidysenteric reputation of this drug has been since its virtue was made known by Piso and Helvetius, in the latter

¹ *Last Mission to Africa*, p. 145.

² Chisholm says (p. 217) that he has known fever cases in which nearly six thousand grains of calomel were required to excite salivation. He did not recommend it in heroic doses in dysentery.

half of the seventeenth century. We find Richard Towne speaking of its use in West Indian dysentery as highly in 1726 as Ewart now does of its employment in India and elsewhere. Amid all the conflict of medical opinion with which the modern history of therapeutics is fraught, I am not aware that any one has objected to ipecacuanha in dysentery; but, except when it was given in ten-grain doses of Dover's powder, on Sir James Macgregor's recommendation, it was rarely so sparingly employed as in 'Twining's pill' (4 to 6 or 8 grains with blue pill), now probably quite out of use, with which I had, in my early Indian career, fair success in some very severe cases.

Dr. Cornish has published¹ a most interesting extract from the records of the Madras Medical Board, giving details of a controversy in which it is shown that, in 1807, Mr. Abercrombie, surgeon of H.M.'s 34th regiment, then stationed at the Hill Fort of Ghooty, indented for six pounds of ipecacuanha powder, which it was his peculiar practice to use largely in dysentery. One pound only having been sent to him he protested that 'it will not last more than three days as I now use it.' In despair at seeing so many of his men die for want of the remedy he had confidence in, he made his complaint to the commander-in-chief. He appears to have used doses of opium 'equivalent to fifteen or eighteen grains,' seemingly in combination with the ipecacuanha. Copland shows (Dict. pp. 731-32) that, in the first and second decades of this century, ipecacuanha was given, with laudanum, in doses of from ten to ninety grains.

In modern times very large doses of ipecacuanha were not used in India until, in 1859, Mr. Edward Scott Docker, of the 5th Fusiliers, introduced a practice which, having been speedily adopted, must have given life and health to thousands of sufferers. For this noble service to medicine and to humanity, Mr. Docker has received, in October 1885, a civil list pension of 100*l.* per annum, announced as being bestowed '*for having discovered,*' [re-introduced], '*a method of treating dysentery by ipecacuanha.*'

I rarely gave this drug in two-scruple or drachm doses after the first dose. More than from fifteen grains to a scruple appeared, in my practice, not to do any harm, but to be a waste of the drug. The means by which an irritable stomach may be made to tolerate ipecacuanha must now be so generally known, that I need not repeat them, save to say that I used opium in moderation, and that, of late years, I always observed Joseph Ewart's rule:—'*It will be found beneficial to time the large doses, so as to allow of one being given night and morning so long as their use is considered necessary.*' The drug is best tolerated by the empty stomach, and the early morning and evening doses are generally slept upon. This plan also has the

¹ *I. M. G.*, for September and October 1869,

great advantage of enabling the patient to take sufficient food during the day.

I had, in my own person, a convincing evidence of the fact that, as a general rule, ipecacuanha is tolerated in dysentery only while the disease needs it. In a rather obstinate attack, my friend Dr. Edward Goodeve desired me to take three-grain doses and to remain in bed. As I lay after my first dose, my mind was full of misgivings; but I did not feel at all nauseated. I went on with the pills, and got well rapidly. Some weeks afterwards, being in good health, I dreamt that I had dysentery, got out of bed, took a three-grain pill, and immediately went to sleep. I was suddenly awakened, and was as sick as if I had taken a full emetic. Imagination could not have been at work here, because I fully believed that I could take the pills with impunity; and, indeed, had no opportunity for thought. The ipecacuanha, being no longer needed, ceased to be tolerated. When I first began to give large ipecacuanha pills, the apothecary made the great mistake of keeping a stock ready. The mucilage drying made them almost as hard and insoluble as shot, and they, of course, passed through the bowels unchanged as irritants.

Morehead laid down the invaluable injunction that: 'When a dysenteric patient is intolerant of ipecacuanha, we should make it a rule to investigate closely the condition of his liver' (p. 388). I fully realised the truth and importance of this observation in my own practice.

A sailor with dysentery in my ward could not be brought to tolerate ipecacuanha, and rapidly sank. On examination, his liver was found to contain distinct traces of numerous old abscesses which had undergone repair. A civilian, aged between 30 and 40 years, a very fine ruddy man, who probably drank beer 'generously,' and whose hand shook, had such very obstinate vomiting from three grain doses of ipecacuanha, in what appeared to be a simple attack of acute dysentery, that I, at one time, thought that I should have to discontinue the drug; but, at last, it was tolerated. He recovered slowly but completely. He went to another station, but died, within two years, of hepatic abscess.

Recumbency must be strictly observed throughout the attack.—I am indebted to Dr. Henry Wilson for a fact which may possibly be well known, but with which I was unacquainted until he mentioned it to me, and which I should certainly have tested in cases where ipecacuan was ill-borne. In indigestion and nausea, it is a mistake to lie on the right side, which brings the gastric contents against the pylorus. I have frequently experienced the immediate relief which turning to the other side effects. In this position, matters find their way to the pylorus when it is prepared to receive them.

Large enemata in dysentery have found many advocates since Dr.

O'Beirne proposed them in 1822. My friend, Mr. Scriven, has given a very interesting review of this practice, to which he has added his own very large experience of its use.¹ Mr. Scriven had, early in his Indian service, full opportunity of seeing this practised, at the time of the second Burmese war, 1852-53, as it was employed by E. Hare, who introduced it in India. Both Hare and Scriven always used it, of course, most cautiously, but quite fearlessly. I may have been cowardly in allowing myself to be deterred by a single unfortunate case; but, knowing how deeply the ulcers often perforate, the following occurrence, which came to my notice many years ago, caused me to avoid a means of treatment which might have benefited many of my chronic cases. A European soldier, having, as was very usual, neglected his illness, applied at an up-country hospital on about the sixth day of his illness. The medical subordinate—a very careful and capable assistant—finding that there was much rectal distress, gave a pint of warm water as an enema. Symptoms of effusion into the peritoneum at once set in, and death speedily followed. I have certainly never heard of any parallel to this case. The astringent which Mr. Scriven has invariably used of late is alum. He finds that the large intestine will hold, in some cases, six imperial pints. As a rule, he orders four pints to be prepared, somewhat warmer than the body, and as much of this to be used as can be introduced without distress to the patient. Although dysentery usually marches side by side with malarial fever, it is not subject to the power of quinine, which, however, ought to be given steadily throughout convalescence.

MALARIAL FEVER:—INTERMITTENT FEVER.

As I have already intimated, I have lived and practised, and shall probably die firm in the belief that intermittents and remittents are caused by a specific poison or morbid entity which emanates from certain soils. I have read, with care, goodwill, and respect, all that has been written by my friends Drs. Oldham and Lyons and others against this view, and I remain fixed in the opinion that heat and chills and damp and sudden and great variation of temperature, are not the primary and true causes of these fevers, any more than the application of a spark is the true cause of the explosion of a loaded gun—these being alike merely the *exciting* causes; the specific poison already in the system, and the presence of the gunpowder in the cartridge, being the only true and valid causes of the discharge of the gun and the outburst of the fever. I have also read all that has been written regarding the *Bacillus malarie* with the greatest interest, and with a wish that my Indian brethren had contributed more to the

¹ *Lancet*, vol. ii, for 1882, p. 259.

practical investigation of this most important point. I can perceive that, should the existence of this microphyte, or of one allied to it, be demonstrated in soils and in the bodies of man and animals wherever fevers of this class prevail, and not generally or largely elsewhere, nearly all believers in paludal poison who may survive when this fact comes to be established will view it as a great discovery which fits accurately into all the four corners of the malaria doctrine of Macculloch.

I commenced this commentary with a promise that, except for the purposes of argument and illustration, I would endeavour to include in my remarks little or nothing which a young English physician should be expected to know upon commencing practice in India. Such a member of our profession, who has passed all his years in malarious Britain, ought generally to have a rather full practical knowledge of paludal fevers; although, in our great home hospitals, however prevalent the masked and hybrid forms of these diseases may be, the common agues of the Kent and Essex marshes and the Lincolnshire fens seldom find admission. Besides this, in his Croonian Lectures, on the 'Climate and Fevers of India,' Sir Joseph Fayrer has given to all students a view of Indian malarious fever so just and so comprehensive as to demand a place among the classics of our literature. Still, the subject now lies before me in the way which I said, more than eight years ago, that I would endeavour to follow to the end, and I think that something to the purpose may be recorded by one who practised among these fevers throughout a working lifetime, who has suffered from one of them for thirty-seven years, and who was scarcely free from an attack at the moment in which he first penned these words.

It has long appeared to me that we ought to possess a work on medicine in which each disease is commented upon by physicians who have been themselves its victims. Acting upon this idea, I shall make this chapter on Indian intermittent fever a kind of running commentary upon my own case of Chittagong fever, which disease affords a tolerably fair type of the intermittent of Lower Bengal.

I need scarcely premise that everywhere, and in all persons, the types of all fevers present infinite varieties. In the lapse of years, in different zones, in dissimilar climates and soils and races, and individuals of the same race, the type of each disease, nay, even the character of each several attack, changes perpetually. In speaking to my students of Indian paludal fevers, cholera, and dysentery, I used to say that I had never in any two years, or even in any one season, seen the same type of disease precisely repeated; nay, further, that, accurately observed, every man's disease differs from those of his neighbours in the two next beds just as much as do the features and figures of the three individuals. While I watched the typhus of London for twelve years, the type of every outbreak was different, often most strikingly

so, and each individual case had its own particular characteristics. I have always borne in mind the observation of an able London practitioner, Mr. Greenwood, which I heard more than forty years ago. It was to this effect. Somebody having remarked that typhus was always adynamic, and could not endure depletion, he said: 'True, as regards the typhus of the present day. I have carefully watched typhus in London for thirty years; during that time my opinions upon this disease have never changed; but, of necessity, my practice has wholly altered to meet the alteration in the character of the disease. Formerly, typhus needed bleeding and leeching, which were undoubtedly useful; but, for many years past, London typhus has been an adynamic disease in which no blood-letting can be borne.' I have no doubt that, if an Essex and a Lancashire practitioner were to change places, each would, if a close observer, find that he had come upon a phase of malarial disease which was new to him. Even in the same individual, the character of each attack is different. Although I have suffered from at least fifty attacks of intermittent, my sensations have, from time to time, varied exceedingly—more than once so much as to lead me into a wrong diagnosis, always to my hurt, and once to my peril. Speaking generally, my full personal experience of Chittagong fever, there and in England, teaches me that the paroxysm is very far more violent in London and in Cheltenham than it is in Eastern Bengal. The tendency to splenic enlargement, as a result of malarial poisoning, varies greatly in different localities. Another out of a thousand illustrations which might be given of the fact that the types of paludal fevers vary with the locality:—generally (in my time—and an observer can only hope to give a true description of the fevers of his own time) it was extremely rarely that the paludal fevers of Bengal produced that blood disintegration which is characterised by a sallow skin. In my chapter on Indian relapsing fever, I have mentioned that, when many Europeans who had been engaged in putting together an iron bridge in a horrible swamp on the railway between Calcutta and the Mutlah came into my wards with remittent fever, a single glance at the complexion of a new patient told me that his was a case of Peealee fever.

With regard to my own case. The first thirty years of my life, although endowed with much good health, were but little conducive to my bodily welfare in India. Until the age of six, I was at my birth-place, Greenhithe, on the Thames, a chalk hill closely advanced upon on three sides by widespread marshes. Many of my father's patients, among the neighbouring poor, suffered most horribly from facial neuralgia, to which he was also subject. The marsh people were often afflicted with 'ague-cake' and many of them were 'liver-grown.' Swancombe, a pretty village on a slight elevation, about a mile inland, was then very unhealthy (it has, within the last few years, been the seat of a remarkable outbreak of diphtheria). My father, who had

served in men-of-war for some years in the West Indies, said that only there and at Swanscombe had he treated severe dysentery. I must have suffered from dysentery repeatedly at Greenhithe; and, there, I had long a sensation, under my left false ribs, which must have been caused by the dragging of an enlarged spleen. Thence we removed to Southsea, which, except on its sea-border, had then, on every side, swamps and marshes, which have now to some degree been improved away and built over. Here I, in a few weeks, had an attack of fever—I know not of what type—which imperilled my life; and, some years later, my father had a return of facial neuralgia. I now became subject to a neurosis—spurious croup. The first twelve years of my professional life were spent chiefly in Maze Pond, Pimlico, and Upper Stamford-street, all originally paludal localities. In the latter street, my father and I both suffered from severe attacks of acute dysentery. I have given these details as some ground for the hint that *the British soldier ought not, if possible, to be recruited in marshy districts*. It has often been stated that Ireland is nearly free from intermittents and remittents, but their congeners, typhus, true enteric fever, and rheumatism, abound. Talking over this subject some time ago with my friend, Dr. John Macpherson, we agreed that at least many parts of Scotland are nearly free from ague, but he added that the ‘shacks’ are not unknown. Until the East India Company ceased to recruit European soldiers, their sole dépôt was at Warley Barracks, now a Queen’s dépôt, and every man was embarked at Tilbury Fort, which lies sunk in the broad marshes of Essex, not far from my native place. In examining a man recently arrived in India, Dr. Macpherson found a huge spleen, and inquired where he had last resided. The reply was, ‘at Tilbury.’ I think that this question of localities to be selected for recruiting, which might be largely illustrated, demands practical attention.

Of the Indian soldier who has been recruited from a paludal district, it may, I consider, be truly said that he, in

‘the moment of his breath
Received the lurking principle of death.’—POPE.

Shortly after my arrival at Calcutta, I had an attack of dysentery, which was easily checked. Several years later, I had two other moderate attacks. Having been stationed at Purulia, Maunbhoom, (a dry station and not very malarious), for a few months, I obtained the medical charge of Chittagong, a place which lies within sight of the sea, at the distance of two miles, as the crow flies, from the mouth of an important river, our houses being built on low sandstone hills, and almost surrounded by alluvial marshy flats of enormous extent, completely inundated during the rains. This place is notorious for its fever. I joined before the setting-in of the rains. A great cyclone had lately devastated the station. The vegetation, which is more

rankly luxuriant there than in any spot with which I am acquainted, had been crushed to pieces by the force of the hurricane. Multitudes of large trees had been either uprooted or broken, and the numerous tanks and ill-dug drains were filled with decomposing vegetable matter. When I arrived, nearly the whole of the residents were down with intermittent fever of no great violence. Although we occupied a house which I afterwards discovered was notorious for its unhealthiness, vast marshes, which extended further than the eye could reach, being open to our view, we appeared to remain well for nearly a twelve-month, during which time, however, paludal cachexia was doubtless establishing itself in our systems. I had started on my round of visits early one morning, and, having seen my dispensary patients, was, on leaving, stopped in the road by a European. As I spoke with him, I experienced an annoyance felt by all who take morning exercise in Bengal—the painful heat and glare of the almost horizontal rays of the sun upon the eye and temple most exposed. I therefore moved into the shade of my buggy hood, where I almost immediately felt chilly. Going home as soon as possible, I had my first attack of Chittagong fever. Neither the cold, the hot, nor the sweating stage was severe, although they were distinctly marked. In all my after attacks of fever in *India*, for eighteen years, there was no proper cold stage—only a moderate sense of chilliness, with malaise. Those fevers of Bengal with which I am acquainted are generally, both in natives and Europeans, unaccompanied by a very marked cold stage, except in the *first* attack. I never saw very severe ague in that country, although the surgeon of a Queen's regiment at the Calcutta General Hospital—a healthy-looking, wiry, middle-aged man—was said to have nearly died in the cold stage of intermittent. Convulsions sometimes take the place of the cold stage in Bengal, especially in children. Ague is much commoner and infinitely more severe in England than in Bengal. This is also the case with retired Indians. After eighteen years' service in India, I had two paroxysms of ague at Cheltenham of most overpowering and terrible violence, the rigors being almost as severe as those which attend the formation of liver-abscess. In my more recent attacks, I have merely felt very cold, but have not shivered.

Soon after my first attack, I began to experience a remarkable phase of Bengal fever. The homeward mail then only left India once a month; and I found that, whenever I had to write my letters, I was seized with fever. This form of periodicity has been frequently observed. My friend, Surgeon-General Cockburn, tells me that he was subject to it when he left Calcutta about six years ago, having contracted it while travelling in Lower Bengal on inspection duty. This '*sol-lunar influence*,' as it was termed, was much discussed early in the century, especially in the writings of Dr. Balfour. I shall revert to it.

The natives of Chittagong, like those of Bengal generally, suffered

from quotidian. In Europeans, the prevalent type was tertian. I never saw a case of distinct quotidian in a European adult. In European children, the fever usually appears to be continued, but may incline to the quotidian type. I never saw a case of tertian in a native. Nor did I ever see quartan in a person of either race. Out of Bengal Proper, these rules do not hold good. I need not follow the subject in its application to those parts of India in which I have not practised. Every medical officer, upon being posted to an Indian station, only too easily discovers the type of its prevailing fever. Seeing that other Europeans always suffered from tertian, I, finding that I could invariably cut my attacks short, after the first paroxysm, by quinine, took it for granted that I was also subject to tertian. This was put to the test, at the end of eighteen years, in Cheltenham. My first paroxysm was of such surprising severity that I did not recognise my ancient enemy, and imagined that I was attacked by some English disease; the fact that I was nearly well on the next day ought to have warned me of my position, but did not. On the third day, the fearful shattering rigor, and the long and violent hot and sweating stages announced unmistakable tertian. This is the only occasion on which I have failed to cut my disease short by quinine after the first paroxysm. I had no attack of croupy cough in India; but, at the commencement of my second cold season at Chittagong, I began to suffer from another and worse neurosis—malarious asthma—a disease which I shall describe presently. This attacked me nearly every cold season during twenty-seven and a half years' Indian service. At Chittagong, I frequently went upon my rounds during the hot and sweating stages of fever. It often struck me that I was more ill than the generality of my patients; but this is evidence that the attacks were not severe. During three-and-a-half years' residence at Chittagong, my family became thoroughly subject to the fever, which killed our eldest child. We who escaped were pale, emaciated, and cachectic. During many subsequent years in Calcutta and its vicinity, I had less fever, but asthma continued in almost unabated severity; and, in the seventh year of my residence in India, I had a very severe attack of acute hepatitis.

Where the intensity of the malarious poison is great, both intermittent and remittent fevers are liable to have their (1) cold, (2) hot, and (3) sweating stages, respectively, most severely complicated by *Pernicious Conditions*—(1) eclamptic, (2) ardent, simulating heat-stroke, (3) 'algide or diaphoretic,' with collapse.

Eclamptic Complication.—Convulsion—rigor being a degree of convulsion—sometimes takes the place of common rigor in the intermittent of Bengal. This is the form of pernicious complication designated by Hertz as the *febris intermittens eclamptica*, or *epileptica*, occurring mostly among children and puerperal women.¹

¹ Ziemssen, vol. ii. p. 608.

This formidable complication occurred in malarious England in old times. Under the date of March 5, 1632, Sir Simonds D'Ewes writes, in his Autobiography: 'I was suddenly sent for by my wife in the afternoon, who had been much affrighted by our little daughter Anne's twice sounding' [swooning] 'away, which, it seems, were fits of convulsion. Before I got home in my coach she was pretty well, but had, after this, so long an ague, being not yet two years old, as we much feared we should have lost her.' Convulsion and epilepsy were very prevalent in this family. Upon my sad Indian experience of this form of disease, I wrote my 'Inquiry into the Circumstances of the Death of King Charles the Second of England,'¹ the King, as I believe, having died of intermittent fever with convulsion assuming a periodic character. Dr. Morehead has given² the case of a man at Bombay who had been admitted with remittent fever of about a week's duration, and who had suffered from previous attacks of fever during four months' residence in Bombay. At midnight of his second day in hospital, he had a convulsive fit passing into coma. There was another fit in an hour. At 2 A.M., there was perfect coma with convulsive movements of the limbs. He expired eleven hours later.

Dr. John Jackson, now residing at Brighton, told me that he had met with this disease in Bengal, occasionally in adults, especially in an apparently healthy young man in whom it proved fatal in three or four days. It is clear that certain of the cases of 'Fever with Epilepsy,' described by Dr. Geddes in his 'Clinical Illustrations of the Diseases of India,' page 259, were of this description. I never chanced to see malarious convulsions in an adult. Dr. Arthur Payne has met with a good deal of this disease in grown-up persons; and, partly at my request, published a very important memoir upon it in the number of the 'Indian Annals' which contains my 'Inquiry.' I saw seven or eight cases of malarial fever, ushered in by convulsions, in children between three and ten years of age, in Bengal. Three proved rapidly fatal; one of these latter, my own first-born, an apparently healthy girl in her fourth year, who had not previously suffered from fever, was, at Chittagong, seized with convulsion, without any premonitory symptom, while playing with her toys. Strong fever ensued. About twenty-three hours from the commencement of the attack, there was again convulsion, in which she died.

Dr. Sullivan says that, in Havana, convulsions are frequent in children suffering from fever, and are perhaps, next to black vomit, the most dangerous symptom. He notices that, when they make their appearance *at the close* of an attack of fever, there is always danger to life. In all my cases, convulsion *commenced* the paroxysm.

Ardent or Heat-stroke Complication.—In the chapter on Ephemeral Fever, Common Continued, and Ardent Fever, I state and illustrate

¹ *I. A.*, No. 14, 1861.

² *Op. cit.* p. 82.

the fact, that, when the hot stage of a paludal fever occurs on a heat-stroke day, the symptoms may be those of insolation. In any case of heat-stroke, we should always endeavour to discover whether the patient is known to have had fever malarial lately or at any previous time, and whether a sense of chill preceded the great heat of skin.

My friend, Surgeon-General Longmore, has given ¹ five cases of men of H.M.'s 20th regiment, who, having been admitted to hospital for remittent fever, were attacked there with symptoms of heat-stroke. Four of these patients died. These cases occurred between May 23 and June 14, when the heat was most intense. I paid my friend a visit at this time, and never felt the heat of India more painfully. There were, altogether, sixteen cases of heat-stroke and 100 of remittent fever in the regiment on that occasion.

Algide Diaphoretic Pernicious Complication, or Collapse.—Dr. Morehead ² relates a case of remittent fever of about four or five days' duration, in which 'the evening febrile exacerbation terminated in unexpected and fatal collapse.' He draws this corollary: 'The period of death, in protracted fatal cases, will not be the exacerbation, but the remission. In describing the sweating stage, Sir Joseph Fayrer has some very important observations upon the tendency to fainting and collapse—as in a striking case which he gives, where the deliquium was suddenly fatal—which is liable to follow a severe paroxysm, especially in the sweating stage. The late Dr. Sutherland gave me the following note: Mr. —, of the Civil Service' [whom I recollect as a flabby-looking man of about fifty years.—N. C.], 'was attacked at Patna, at the end of the second week of June 1857, with a low form of remittent fever. The case became serious. He had a very weak pulse, and was suspected to have a fatty heart. He was attacked in the Opium Factory, which was fortified against the insurgents, to which he had come as a place of safety. After a few days, it was decided to take him back to his own house at Bankipore. Here he imprudently took solid animal food, unknown to Dr. Sutherland. The consequence was a violent attack of diarrhoea, which, for a time, caused anxiety with regard to the result. Later, the case appeared to go on favourably, and a senior medical man who saw him, in reply to his anxious desire to have a bath, prudently said that he might be sponged with warm water. On the evening before his death (on or about the tenth day of his illness), Dr. Sutherland found him in apparently a very improved and satisfactory condition, and he was considered to be out of all danger. About seven o'clock on the following morning, Dr. Sutherland was hastily summoned, and found him in a state of complete collapse, and quite unable to give any account of himself. He was cold, and drenched in perspiration, which wetted the sheet on which he lay. The pulse at the wrist was scarcely perceptible. Dr. Sutherland was

¹ *I. A.*, No. 12 for 1860, p. 396.

² *Op. cit.* p. 69.

able to administer stimulants, and to use heat and friction, and to apply sinapisms to the legs, &c., but he rapidly sank. On inquiry, it was found that he had been so imprudent as to allow his bearer to sponge him before an open door in an outer room communicating with a verandah. The morning was rainy and windy [the rains must have set in a day or two previously ; then, the sudden change to chilliness from extreme heat is always most trying to the sick.—N. C.]. It was during or immediately after the act of sponging that collapse came on.

This was evidently a death from algide pernicious fever, or what Hertz terms *Febris perniciosa diaphoretica*. Mere syncope, which may, however, be attended with great danger, is apt to occur in the sweating stage. Once at Chittagong, having an unusually severe attack of intermittent fever, I fell asleep as sweating commenced, and dreamt that I was out in the Bay of Bengal, lying on the thatch of a native boat, with a cold sea breeze passing over my body. Waking, I found that my hair was as wet with perspiration as if it had been dipped in a bucket of water. I got out of bed, and went into the bath-room, when it occurred to me, 'Something has happened !' Presently I found myself flat on my back, recovering from a 'dead' faint. That which 'had happened' was evidently the striking of my occiput on the terraced floor. In fainting at other times, I have always had some little warning. The same accident happened to a middle-aged man whom I attended in Calcutta. In falling, he cut his brow severely. I saw this symptom, in a case of Indian fever, a few weeks before this chapter was first written. Where the heart is weak or the cerebral arteries are faulty, circulation may fail to be restored ; but I do not think that mere syncope, in or after the sweating stage, is to be regarded as evidence of perniciousness.

I have seen diarrhœa and, as I believe, profuse bronchial defluxion replace the sweating stage. Vomiting usually occurs in the hot stage ; and when it does not, it should be induced by ipecacuan and tepid draughts, as it is nearly always the signal of commencing perspiration. Sweating generally commences first over the mastoid processes and on the inside of the wrists. When, in the absence of a paroxysm, I found that this latter surface was moist and clammy to the touch, I always knew that fever would return if not arrested by quinine. When the skin at this spot felt cool and pliable, exactly like the leather of a soft and new white kid glove, I knew that the attack was at an end. These signs never led me into error. I have never seen an exception to this rule. When a paroxysm returns earlier than the immediately preceding one, we may be sure that the disease remains unchecked ; but, whenever it comes later, we may be confident that we are subduing the fever.

Fayrer, citing Maclean, says that 'the direct mortality from intermittent fever in India is small. In Bengal, out of a strength of 344,152

with 111,687 admissions, the percentage of deaths to strength was 0·24, of deaths to admissions, 0·76.'

Treatment.—I seldom found occasion to interfere in the hot stage, except when headache and fever were very obstinate. Then I kept the head cool and excited vomiting by ipecacuanha. I *once* applied a few leeches to the temples, without advantage, in the case of a man whose headache was very severe. *Immediately* moisture began to appear, in the manner noted above, my assistants were directed to give a moderate purgative. This was generally retained; but, if not, vomiting was followed by sweating, and the dose was repeated. When the cinchona bark was first brought into use in England, all writers insisted that, to render it efficacious in ague, the first passages must be cleared by a purgative. Even Dr. Salmon's 'Bates's Dispensatory' (1694) enjoins this precaution, which must never be neglected in India. When the purgative was retained, I gave six grains of quinine to an adult, without waiting until general perspiration set in. The steady use of quinine *during the sweating stage* moderates the severity of this crisis, and probably defends the patient against syncope. Many of my professional brethren value large doses of quinine. I do not; they often cause terrible headache, and do not appear to be more efficacious than moderate ones. My [practice for many years scarcely varied. In the case of every adult European male, I ordered half a drachm of quinine in ten three-grain pills—two to be given on the first appearance of perspiration, one every three hours during the interval, and two at the *very slightest suspicion*, right or wrong, of the recurrence of a paroxysm. I used to tell my students that the European male generally required thirty grains, the European woman and the native male from twenty to twenty-five grains, and the native female from fifteen grains to a scruple. I found this law to be very constant.

For some time before I left Calcutta, ten years ago, the native public had almost utterly lost faith in the efficacy of quinine, and I had great difficulty in persuading several of my native professional brethren, the leading practitioners, to prescribe it. Doubtless this scepticism originated with the non-professional native public, who, seeing that quinine often failed to cure Burdwan fever (it will not cure starved people who get it too late), considered it powerless. It was believed also that there was a great deal of spurious trash, falsely called quinine, in the Calcutta market. I, at last, began to fear that the inestimable blessing of the great anti-periodic was about to be lost to malaria-impested India. More than ten years ago, I observed a fact which may have partly afforded ground for this incredulity. I met with cases of what appeared to be ordinary intermittent that had been neglected, which resisted the power of quinine with extraordinary persistence. We always anticipated such difficulty in ill-fed natives with long-standing malarious cachexia and splenic disease, but I am speaking of what

seemed to be simple and uncomplicated cases. I especially noted the cases of a mother and daughter occupying beds in my native ward, who I fully expected would recover in from three to five days. Although I poured in quinine steadily and freely, the paroxysms recurred daily for eighteen and twenty days. It appeared to me, latterly, that nearly all cases of malarious fever displayed much of this stubbornness of resistance against the specific.

Whenever the inexperienced practitioner finds that a remedy of generally acknowledged efficacy fails in his hands, he may take encouragement in recollecting that there is great uncertainty in the 'certainties' of Nature, especially when man's attempts to bring them into operation are hasty or clumsy. Thus, fire has usually the power of exerting very strong action upon paper and chips; but every shivering retired old Indian knows how very difficult it often is to light a fire. The materials are not properly laid, or they are damp, or there is not enough of them, or the register is shut, or the doors or windows are closed. So quinine is the true specific antidote to paludal fever, extinguishing it as certainly as water quenches fire, and as fire ignites wood, but it often fails to act as such. It may be impure, or may be injudiciously administered, as where the bowels are allowed to remain loaded; or it may be given in too small or too large doses; or we may not have a chance of employing it until the patient is moribund beyond recall; or the patient may not be properly fed, or clothed, or lodged; or there may be old organic disease. We must not expect quinine to do the impossible; and, in using it, we must not only have faith in its power, but we must, as in conducting any other delicate operation, surround the process of its employment with every precaution needful to secure its effectual action. To cite a single example: I was told by a retired brother officer of great experience, chiefly in the North-Western Provinces, that a common cause of remittent fever is neglect in the precaution, which he always employed, of wrapping the patients in blankets during the sweating stage of intermittent. This was not my practice, and I was not convinced that my friend's theory of the causation of remittent fever is correct. Still, there can be no question that a chill, during the sweating stage, is dangerous in the extreme, and may render our quinine treatment abortive.

A minor difficulty, in regard to quinine, has probably always beset the physician in India. Among European ladies and gentlemen, about one patient in five says, very impressively, at the outset of the case, 'I have not been able to take quinine for years, it always disagrees with me; you must not give me a grain!' My first Chittagong fever case, the collector, a corpulent, middle-aged gentleman, very ill, gave me this preliminary warning. I listened civilly and silently, thinking: 'If you really cannot bear it, you will probably die.' I sent him a full supply of the alkaloid, and watched its action carefully. It did not

produce a single unpleasant symptom ; it at once commanded the fever ; he recovered quickly, and when I last saw him, two or three years ago, he was a remarkably well-preserved old man.

Having always used quinine freely, but carefully, and in moderate doses, I have never seen it do the slightest harm, nor can I accuse it of causing permanent deafness or any other ill result. Early in my practice at Chittagong, I was treating a young infant for the diarrhoea which is prevalent among teething children in India. Having ordered moderate doses of quinine, I found, to my horror, that, by my dispenser's error, the baby was taking what I feared must be poisonous doses. I watched the case in great suspense for many hours, but not the slightest evidence of increased bowel-irritation nor any other evil result was perceptible.

I was more than once desired by the head of my department to try, and to report upon, the cinchona alkaloids and other preparations of bark. I found them all very efficacious in common cases of intermittent, but I know nothing of their power in grave cases of remittent ; as, having quinine at hand, I would never use them when life was in peril. I sometimes wished that I had, for a short time, been so placed as to be compelled, in the absence of quinine, to test their efficacy in serious cases. Certain it is that the true measure of their power ought to be clearly ascertained. In dispensary and native military hospital practice, atees (*Aconitum heterophyllum*) of the lower spurs of the Himalayas, and powdered kat kaliya (*Cæsalpinia Bonducella*), the pods of which may be seen growing in every swamp in Bengal, washed down with infusion of chiretta, are most useful in simple cases. On one occasion, as I have already stated, my supply of quinine and cinchona bark having failed, ordinary intermittent being very prevalent in my jail and sepoy hospitals, I had nothing but kat-kaliya and chiretta for six weeks and found the mixture perfectly efficacious.

Although quinine and other tonics may, by maintaining constitutional power, render persons residing or travelling in malarious districts capable of resisting fever, and go far towards mitigating the violence of its attacks, all experience combines to prove that quinine is not a valid prophylactic against severe intermittent. Thus, if I get fever to-day, and at once check it by taking half a drachm of quinine in divided doses, the daily use of quinine, in such doses as I could bear and work under, will not prevent me from getting a return of fever a month hence.

I was taught, and myself taught for many years in India, that, whenever we begin to find that a case of any fever, or dysentery, or diarrhoea, or any other allied malady, begins to resist treatment, REMOVAL must be tried. As, when a man falls into a tank, he must be immediately taken out, so when a person falls ill, especially with malarious disease, in any locality, he must never be allowed to die there. I invariably endeavoured to move such cases from one room to another,

from one house to another, from one street to another, from one end of the town to another ; always, if possible, to a place better in its sanitary surroundings ; but, if not, *anywhere*. A similar law is observed here in England, where cases of chronic whooping-cough, bronchitis, and hay fever are sent to the seaside. Three times, when living in and near Calcutta, I had, personally, recourse to this measure—twice in the case of a member of my family seriously ill with fever, and on the other occasion when I considered that I was dying with symptoms of hepatic abscess. We were carried from our own airy and perfectly well-appointed bed-rooms to a Calcutta hotel, not at all noted for its salubrity, but where all the arrangements were comfortable (it used to be called ‘Chevers’s Sanitarium’). I have alluded to the first case in my note on Denguis—recovery commenced immediately upon removal. The second was a very serious case of remittent fever with persistent head-symptoms. Convalescence set in about twenty-four hours after removal. In my own case, I had been ill in bed for more than a month. I had almost lost the power of taking food ; I was drenched in cold sweat, and I believed that I was moribund. I was carried away to the hotel one evening ; I slept comfortably all night, and, next morning, I had a keen appetite, and felt that health was returning. No symptom remained but considerable debility. The same observation has been made in regard to yellow fever in the West Indies, and, I believe, to typhus in England.

There can be no question that, as a general rule, the constitution of victims of paludal cachexia is benefited by removal to a pure air in a non-malarious locality. Thus malarious asthma, which never entirely ceased to persecute me in India, has hitherto, in more than twelve years, wholly spared me in England ; but the scope of this rule is greatly qualified by the law, recognised by every Indian practitioner, which the following cases illustrate. A young lady who, after many months’ residence at Chittagong, appeared to have escaped fever, went with her father, our chaplain, to Tipperah, which was not then considered as a very malarious station. She was immediately attacked with intermittent fever. A medical officer had never suffered from fever in India. He was sent to England for dysentery. He recovered from that disease ; but, throughout his three years’ sick furlough, intermittent fever persecuted him incessantly. Sir Ranald Martin, who attained advanced old age, and who remained generally healthy for some thirty years after he left India, told me, about a year before his death, that a fever which attacked him in the Cuttack Hills, when he was a young officer, had never since failed to persecute him every spring. Again, to refer to our own cases, two of us, retired from India for six and five years, are now getting far more severe paroxysms of intermittent in Bayswater than we ever had in Chittagong. [This was stated four years ago ; we have since happily escaped.]

We used to observe the operation of a similar law in England before I went to India, in English ague and in other diseases. A city man would be treated for primary sore, and be recommended to take change of air, say at Hampstead or the then open Surrey Hills. In a day or two, a papular eruption would make its appearance.

In cases where the paroxysm is ushered in by convulsion, the bowels should be freely cleared by a calomel purge and a cathartic enema, and quinine must be given early and freely.

Knowing that quinine does not cause bowel-irritation, which is always to be dreaded in India, and having full confidence in this specific, I never prescribed arsenic in fever, and very rarely employed it in other diseases.

ENDEMIC MALARIAL REMITTENT OF THE PLAINS.

Examination of the recent statistics of Indian fevers, given by Sir Joseph Fayrer and others, proves that (1) the mortality from true enteric fever in that country is by no means extremely large; but that (2) the death-rate from malarial remittent among the civil population, although it has not been precisely defined, is probably enormous.

(1) Sir Joseph Fayrer shows that, in 1879, (when a great fever epidemic swept over India) the British army stationed in that country had 387 fever deaths out of a strength of 57,810. These deaths cannot all have resulted from enteric fever. The same authority gives a table in which the enteric fever cases treated in the Calcutta Medical College Hospital, in the ten years 1871-80, appear to have been 3, 5, 9, 5, 6, 6, 1, 0, 2, 0. Dr. Vandyke Carter mentions, in his work on 'Spirillum Fever in Bombay,' p. 438, that, in seventy-four autopsies of native patients dying from 'remittent fever' during 1877-78, intestinal lesions like those of typhoid were found four times. He evidently considers that these four were probably cases of enteric fever.

(2) We have already seen¹ that the direct mortality from intermittent fever in Bengal is small, the percentage being to strength 0.24, and to admissions 0.76. Further, when it is borne in mind that relapsing fever does not occur frequently in India, we may form some idea of the destructiveness of malarial remittent from Sir Joseph Fayrer's report, that, in 1879, the registered deaths from fever in India were 3,564,035, out of a total mortality of 4,975,042, in a population of 187,105,833.

Malarial remittent is prevalent, endemically, throughout extensive regions in the vast plains of India, but its reign is not so almost universal as that of intermittent is. The remittent varies considerably in type in different localities; and history shows that, in the same locality, the

¹ P. 68, and Fayrer, p. 81.

prevalence, type, and destructiveness of the endemic fever have great fluctuations and marked changes. Every physician who has both worked long and read much in India, has probably asked himself, 'Are these fevers which come before me the diseases of which my predecessors have left record?' And everyone of large Indian experience must have remarked great alterations in the character of fevers within his own time. These changes in type often depend mainly upon season and food-supply, and upon inundation and drought, upon the presence of irrigation canals and railway embankments, intercepting natural drainage, and also upon other less explicable modifying influences. With regard to food, it is always to be borne in mind that, apart from the visible destructiveness of great famines, the mass of people—the two hundred and fifty odd millions—are ill-lodged, and are more or less everywhere restricted to food so poor in nutritive elements that they are the subjects of lifelong starvation.

Malarial remittent has its greatest prevalence after the rains, in the dry and cold season, when tanks and watercourses dry up, when the leaves fall and deciduous vegetation rots, and when the ill-clad native poor suffer from cold, which, next to malaria, is their greatest climatic evil. In some years, and occasionally during a course of years, a local remittent appears on so large a scale, and assumes so deadly a character, that even medical men are led to imagine that it has become epidemic. It is clear that the endemic cholera of Bengal does, at rare intervals, become epidemic. The question is, of course, open to discussion; but my own opinion, formed upon long and careful observation, is that malarial remittent, however wide the extent of its ravages may be, is never truly epidemic. Thus it was observed that the Burdwan fever spread remarkably northward of Calcutta, involving localities which had been, of late years, considered fairly exempt from destructive remittent. It had not, when I left India, extended to Calcutta. We used to expect it there; but, if it had appeared amongst us, I should have said that its spread had gained a new increase, but should have denied that it was epidemic.

Considering that medical officers serving in India are nearly always liable to change of station, and that the type of fever differs greatly in various localities, and in the same locality at various times, the young officer ought always, upon taking up a new post, to become a learner, by making full inquiry of brother officers and by reading, especially by studying the reports of his predecessors in his office books, &c. It is noticeable that one great characteristic marks the writing of Indian physicians, past and present—this is, *fidelity of description*—nearly every author doing what every writer on fever should endeavour to do, i.e., stating what his fields of observation were, giving the dates and describing accurately the fevers which then prevailed in those particular localities. On this ground, I would advise the young Indian practitioner

to read all works on the diseases of that country ; but on no account to fail to master the contents of those which describe the maladies of his own locality—Morehead, V. Carter, and Moore for Western India ; Annesley, Geddes, Maclean, Waring, and Francis Day for Madras ; Kenneth Mackinnon for the North-Western Provinces ; James Johnson, Twining, and Ranald Martin for Bengal Proper in old times, and Fayrer and Ewart for the diseases of that province in the present day.¹

Multitudes of cases of remittent fever in India are not brought under notice in their early stages, and, when the disease is seen at its first onset, it is treated so promptly by quinine that it has usually no opportunity of developing itself sufficiently to display its full character for observation. The cities and large civil stations, in the present day, mostly enjoy a considerable immunity from severe remittent, by reason of their tolerably good sanitary arrangements ; and, when native cases

¹ Between the years 1842 and 1844, the Medical Board of Madras caused to be compiled from their records and published a series of Topographical Reports upon the stations of that Presidency. The three volumes which lie before me as I write form a noble monument of judgment and accuracy. The medical history and health statistics of each station are given briefly, but with sufficient fulness. I often wished and argued that, after this manner, a manual should be prepared for every military and civil station in British India as a guide to each local medical officer and as a basis for his reports. Indeed, I attempted something of the kind in my work on 'The Means of Preserving the Health of European Soldiers in India.' It is clear that these manuals should contain the fullest possible details, and should be frequently revised as new facts come in. As regards fevers, they should contain a sufficiently full geographical and geological account of each district by officers of the Geological Survey ; a clear exposition of the habits, mode of cultivating the land, and food of the people ; a history of all recorded epidemics and endemics ; a minute description and localisation of the prevailing fevers in all their types, their statistics, therapeutics, &c. The Government of India did, at one time, encourage the preparation of such district manuals by local medical officers. The result was the publication of about half a dozen singularly valuable works, such as those on Dacca, by Dr. Taylor ; on Upper Scinde, by Dr. Kinloch Kirk ; on the Valley of the Indus, by Dr. Percival Lord ; on Calcutta, by Drs. Duncan Stewart and Strong ; on Oudh, by Dr. Donald Butter ; on Ajmeer, by Dr. R. H. Irvine, &c. ; but the Bengal series was never completed. Many valuable Topographical Reports of stations and districts in the three presidencies of India, such as those published in the *Bombay Medical and Physical Transactions* for 1857-58, have appeared during the last forty years in the medical periodicals of that country. The first compilation of a full series of these manuals, by a few selected medical officers of each presidency under a director, would cost the Government a good deal of money ; but, assuredly, this outlay would be speedily repaid with interest in the improved health and efficiency of officers and men and of the native millions inhabiting the districts. A like plan might, with very great advantage, be adopted in the United Kingdom, in all our colonies, and throughout Europe and America. I made a scheme for the publication of Medico-Topographical Reports for all towns and districts in the United Kingdom and elsewhere the subject of my inaugural address at the Epidemiological Society at the opening of the session 1883-84. (*Vide* the Transactions for that year, p. 1.)

are brought in for treatment, they generally come from remote districts, whence they have only been removed as a forlorn hope when the disease has become very far advanced. Indeed, the majority of native cases are brought in moribund, worn out almost equally by the gravity of the disease, by want of medicine and proper nourishment, and by the fatigue of the journey. Medical officers see most of this disease in native and European regiments, especially when on field service in malarious localities; in officers who have been brought in, immediately upon being attacked, while on district duty near the stations, and in European tea-planters; and, not very unfrequently, in their own families and persons.

The questions—How far are intermittents and remittents allied in etiology? and, Is one of these types of fever convertible into the other? are, although very interesting, of no great practical importance, except to those who consider that malarial remittent fever is either typhus, enteric, or relapsing fever. My own opinion, founded upon much observation and thought, is that they are somewhat cognate but perfectly distinct maladies, caused by different kinds, degrees, or forces of malaria, not always prevailing in the same district, and generally appearing at different seasons of the year—the intermittent being a disease of swamps and marshes, the typical remittent a malady of jungles and terais. In many parts of India, as at Chittagong, where swamp and jungle meet, where marsh extends nearly to the base of thickly-wooded hills, the distinction, of course, becomes rather confused. I believe, almost to certainty, that intermittents and remittents are not degrees or developments of the same malady, or the results of one and the same cause. This question ought to be sifted by those who are engaged in investigating the habits of the *bacillus malariae* in and out of the human body.

I have often speculated upon the probable issue of an entirely neglected case of severe intermittent. What would be its course and development? How and when would the patient die? To me this point is not clear;—even the most ignorant native practitioners have tolerably efficient remedies for intermittent, and the poorest cooly is seldom wholly unattended to in this disease. It is possible that, if the disease were left to its own course for many days in a very malarious district, the intermissions would daily become shorter (we always notice this when intermittent is unchecked), and we should, at length, have a very grave fever very like remittent, but probably not identical with it—rather what old authors termed a ‘typhoid’ or typhus-like condition.

All the reliable facts which I was able to obtain combined to show that the *Burdwan fever* originated as a simple malarial fever, quite amenable to quinine and ordinary treatment when taken in time; but that, in the absence of treatment, poverty and neglect were mainly

operative in leading to the heavy mortality which characterised the disease.

Very lately¹ we have accounts which (although the precise nature of the disease is not perfectly defined) appear to show that, among a barbarous and extremely impoverished race, unacquainted with medicine, and living very remote from European aid, a common intermittent, breaking out at an unfavourable season, may become very destructive to life. There are reports by Dr. Clarke, late sanitary commissioner of Assam, and by Dr. Eteson, his successor, which show, in the words of the latter officer, that 'the deaths in some of the *thanas*' [of the Garo Hills, from *kala azar*, or black death] 'have been enormous.' Little is known of this disease, but it is said to be 'a marsh ague intensified, through chronic lapses, into a blood taint which passes into anæmia and organic congestions' [congestion of organs?], 'and it is only specially fatal because the sick among the Garos are left uncared for, unnourished, and exposed to frequent relapses.' 'They believe in incantations, and will not take European medicines; they cannot be persuaded to undergo hospital treatment as in-patients, and they live surrounded by every conceivable insanitary condition, in a country which, for the greater part of the year, is practically inaccessible. To add to the difficulty, the fever is generally of a lingering character, the sick dying ultimately from blood degradation.'

I have already mentioned the opinion of a brother officer of large experience in the North-Western Provinces, that, if sweating be checked in intermittent, the case is likely to assume the remittent type. Upon this point I cannot speak from experience, except that I have never seen such an occurrence in Bengal. In the paroxysm, the native of that part of India always wraps himself completely in his blanket, head and all, as closely as a corpse in its winding-sheet, so that he is not likely to be chilled; and the European is usually well protected against draughts. Jungle or terai fever stands absolutely apart from intermittent fever; and a leading characteristic of the remittent of Bengal, as I saw it, was that the fever was remittent from first to last. We used to hear, from writers of good authority, of remittents which began and ended as intermittents. But here it is to be borne in mind—(1) that remittents vary much in type, and (2) that the unusual prevalence of intermittent has generally been noticed as a precursor of severe Indian epidemics and endemics, as of relapsing and true enteric fever; and that these are liable to have their types considerably masked and modified by evidences of paludal influence. My friend Mr. Scriven, who first observed and described true enteric fever in India, and who does not hold with the idea that remittent fever is either typhus or enteric fever, notices that 'persons weakened

¹ *I. M. G.* for March 1885.

by malarious fever are particularly liable to typhoid' (true enteric). 'Intermittent also often supervenes during convalescence from typhoid. Enlarged spleen is common to both diseases.'¹ Thousands in Lower Bengal have their systems saturated to the very uttermost with the poison of intermittent, but never become the subjects of remittent. On the other hand, intermittent is no safeguard against remittent. When a denizen of the marsh ventures into the jungle, he may get remittent, and his disease, carefully observed, will probably be found to have some resemblance to intermittent. I repeat, remittent fever is not, in my opinion, modified or aggravated intermittent.

While the duration of those fevers, the course of which is not arrested by treatment, such as typhus, true enteric, and relapsing, has been clearly ascertained, it is more difficult to fix the natural limit of remittent, which nearly everyone, in all times, has essayed to cut short and 'cure.' Dr. Eyre, however, gave² a table from which he inferred that the average duration of remittent is seven days, and that it extends occasionally, but rarely, to fourteen. This, he added, agrees with what is stated by Dr. J. Browne in the 'Cyclopædia of Practical Medicine.' 'Remittent fevers in warm climates continue till about the seventh day; but, in other cases, till the fourteenth. In temperate climates, forty or fifty days or more.'

Judging by many neglected cases which have been brought to me from distant country places, I think that the course of remittent fever, when unchecked by treatment, to a fatal termination, ranges from three days, in the most destructive types, to about eighteen days, according to the strength of the patient, his food, lodging, and other surroundings.

When recovery takes place in neglected cases, it is always slow and difficult, nearly as in English typhus.

The general course of malarious remittent in the plains of Bengal is that it is ushered in by chill followed by high fever, in which, unlike enteric fever, the temperature reaches its maximum in a few hours.

A remission of varying duration, from hours to minutes, in which, in cases of the mildest type and at the commencement of the attack, the temperature is lowered, the head-symptoms abate, and the skin becomes more or less moist, is to be anticipated generally towards morning. But, in grave types of the disease and in its advanced stages, where its course has gone on unchecked by treatment, the remission becomes almost daily less and less distinct, and sometimes is scarcely recognisable by means of the thermometer. There is severe headache, which, if the case be neglected, is followed by coma. There is less frequently delirium. The tongue, teeth, and lips become

¹ *Transactions of the International Medical Congress of 1881*, vol. ii. p. 537.

² *I. A.*, No. 8, p. 539.

covered by sordes. The bowels are generally constipated at first, until, if their accumulated contents are not early removed by aperients, intestinal irritation and diarrhœa set in. Old writers speak much of dysentery as a sequel of remittent; but it is to be doubted whether these were not cases of true enteric fever. As I have already noticed, much of our earliest information regarding Indian remittents was given by ships' surgeons, from Clark to James Johnson, whose men were generally scorbutic. As I have also mentioned, the taint of land-scurvy is a very widely-spread evil in India, especially among the ill-fed poor; but many in good circumstances are, to a greater or less degree, thus affected. This is a point which requires rigid investigation in every case of severe fever. Every young Indian physician ought to study Twining's description of the congestive remittent which, in his time, prevailed during the cold season in Bengal. It is evidently full of sound and careful observation. In receiving and treating the corresponding cases, which crowded my wards every cold season in Calcutta, I do not think I ever saw the precise types of disease which prevailed in Twining's time; but, almost in every case, there were points which brought the name of 'Twining' before me. Among the starved natives, pinched with cold, I especially used to notice the head complication, and the dried-up skin upon which one could write with one's finger-nail. I saw far less of bilious vomiting and purging and of serious abdominal congestion than Twining did. The discoloration of surface from blood disintegration—not that which accompanies relapsing fever—was rarely noticed, but was a marked characteristic of the cases of Peealee remittent, alluded to at p. 106. Dr. Partridge, an officer of large experience in Assam, cited by Fayrer, says that jaundice is no uncommon accompaniment of remittent in that locality, either as the result of mechanical obstruction of the ducts, or of arrested function. In my experience, there was more of coma than of muttering delirium. In the hot season, 'ardency' and delirium occurred but rarely in Europeans, and were easily commanded. Of course, cases of remittent vary much in intensity and in amenability to treatment, but the Bengal remittent, with which I am familiar, was generally a fever of great severity. I know little or nothing of the mild remittents spoken of by my predecessors in India.

In some cold seasons, parotid swelling, always confined to one side, with deep suppuration, was common among poorly-fed natives late in the course of remittent fever. I never saw other glands similarly affected.

PERNICIOUS TYPES OF MALARIAL REMITTENT FEVER.

Algide and Diaphoretic Remittent Fever.—I have already¹ stated and illustrated the fact that Indian remittent has its algide and diaphoretic pernicious types. The cold and sweating stages of ordinary malarious fevers vary much in intensity; but, whenever there is a distinct tendency to collapse in either of these stages, the type of fever must be regarded as pernicious in a greater or less degree.

In his report of cases treated at the General Hospital, Calcutta, for April 1884,² Mr. Raleigh stated that there had been 'a tendency to sudden collapse in some cases, proceeding to an alarming extent, from which, however, the patients rallied.' Thus, in a man admitted on April 13 with 'inflammatory fever,' determination to the head being the most marked local symptom, 'general bleeding, calomel purgatives, and cold applications to the head were directed.' On the 14th, the head was perfectly relieved, and he was tolerably free from fever through the morning, but, at noon, had a regular fit of ague—cold, hot, and sweating stages, succeeded by coldness of the skin and small pulse. Five grains of quinine every four hours. 15th: Paroxysm at noon, which left him without uneasiness. 16th: At the morning visit, he was found in a state of collapse; the skin cold, wet, and blue; pulse feeble and rapid, with oppression at the præcordia and restlessness; the tongue foul. He took a scruple of calomel. The collapse lasted until the evening of the following day, when the report was, 'He is restless; has oppression of breathing; skin continues very cold, and pulse feeble. Has had no stool; belly distended. On the morning of the 18th, the skin was warmer, and he was altogether better. On the 20th, his gums were affected by mercury. By the 23rd, he was reported convalescent. Mr. Raleigh added that 'in some patients despondency, and sensation of sinking and debility, with a quick though weak pulse, prevailed from the first—here local bleeding by leeches was substituted for the lancet when determination to a particular organ was indicated.'

In the following month (May), Mr. Raleigh reported that 'at the latter half of the month, when the heat of the weather became intense, the thermometer standing at 96° in the coolest houses in the day, fevers' [which early in the month had the common inflammatory character, and were easily subdued by antiphlogistic remedies] 'assumed a more malignant aspect; the inflammatory and congestive indications continuing long unabated, and, after many days' duration, declining into a low typhoid³ state, in which despondency, appre-

¹ In the chapter on Intermittent Fever, pp. 167 *et seq.*

² *I. M. J.*, vol. i. p. 207.

³ By this term, the Indian writers of that day meant typhus-like.

hension of death, irregularity of circulation, extreme prostration of strength, with a dry dark furred tongue, frequent small pulse, hot clammy and often yellow skin, and depraved or arrested secretions, were among the most alarming symptoms. Relapses were frequent and repeated, and convalescence tardy.' It is noteworthy that, at this time, relapsing fever was prevalent up-country—in Bareilly, Rohilkund, and the districts around Delhi.

Speaking of the fevers of July, Mr. Raleigh wrote that few of the cases observed the *regular* form of remittent fever; but had, for the most part, been continued fever with exacerbations at given periods, usually towards evening. In the early stage, the inflammatory symptoms commonly ran high, with marked determination to the head. If not subdued at this stage, the disease passed into a low typhoid condition. In cases proving fatal, the patients usually became comatose, and sank gradually. Mr. Raleigh adds: 'Sudden collapse after a paroxysm was not unfrequent, and showed the necessity of strict adaptation of remedies to immediately prevailing states of the system, particularly with reference to general and local bleeding—which, however beneficial whilst a state of inflammatory excitement existed, would probably, if resorted to at too late a period of the disease, or on the decline of a paroxysm, have proved singularly injurious.'

In the autumn of the following year, 1835, Mr. Raleigh had a case which might, at first sight, appear to be of a type intermediate between pernicious remittent fever and cholera, but which, I believe, was not so. A European soldier obtained his discharge at Hazareebaugh, and walked down to Calcutta (a terrific journey at that hot season), and then went about his business in the town for three days. He was admitted on September 26. He was found to be in strong fever, with a deep yellow suffusion of the conjunctivæ and violent headache. He was treated antiphlogistically with bleeding, leeching, and calomel, and did very badly until the evening of the sixth day in hospital, the disease having a paroxysmal remittent character, with severe head symptoms during the paroxysms. On the 29th, he had 'several pitchy stools' in the morning, and 'several green, watery stools in the afternoon, after salts and senna.' On the 30th, he had salts and senna twice, and was 'freely purged—the evacuation a bright green fluid.' On the afternoon of October 1, 'had a short imperfect paroxysm at 2 P.M.; after its subsidence, has been much purged, and is now greatly exhausted. A cold, clammy sweat on the surface, but the intellect is clear.' On the following morning, the report was, 'Slept well and refreshingly; feels much better this morning.' He remained free from fever, and appears to have made a good recovery. Although, in his remarks on this case, Mr. Raleigh speaks of the 'risk of collapse from purging at the decline of a paroxysm,' he also insists

upon 'the necessity of calomel and brisk purgation until healthy secretions are procured.' True, this man might, had his constitution been feeble, have died in the collapse which followed the great purging; but I believe that there was no perniciousness in this purging and the very temporary prostration which followed it. At Chittagong, I attended an officer's child, about seven years old, with remittent fever. She was quite comatose for more than forty-eight hours, during which time her father and I scarcely left her. The bowels were obstinately constipated, and I gave calomel and croton oil with butter placed on her tongue. At length, suddenly, the bed was literally swamped by a great dejection of thin, greenish fluid, upon which, almost instantaneously, the skin became cool and the intellect clear. She recovered without a bad symptom.

These two cases, I repeat, were not, in my opinion, pernicious (the soldier may have suffered from relapsing fever); but it is to be remarked that the really pernicious remittent outbreak in Calcutta, in May, June, and July 1834, as described by Mr. Raleigh, was followed, in September and October 1835, at Deesa, in the Bombay Presidency, by a closely similar malady, which, in some of the cases, assumed the pernicious cholera form. A very full account of this remarkable type of fever is given by Dr. Jackson, H.M.'s 6th Foot.¹

In the month of August, common remittent and intermittent were very prevalent, but there were no casualties. Early in September, the fever displayed a more serious aspect. Although the disease often assumed a quotidian and tertian type, the symptoms were so violent and anomalous that the three stages of an intermittent were lost sight of. In some cases, the disease was ushered in by stupor, syncope, or loss of sense and motion. When the accession was sudden and without premonitory symptoms, the patient had distracting pains in the head, back, and lower limbs. At one time, there was an indescribable sense of oppression on the brain; at another, the patient fell as if he were drunk. Violent pain in the ear, the eyebrow, or the eyeballs, difficult micturition, hemicrania, vertigo, and confusion of ideas were observed. When the chest was principally affected, the accession resembled a fit of asthma: the patient was seized with sudden dyspnoea, gasping for breath. Sometimes there was pain and an awful sense of oppression across the chest, including the hypochondriac and hypogastric regions. In other cases, there was abdominal complication—darting lancinating pains in the epigastrium, tenesmus, and umbilical griping pains; a sensation as if the whole body were distended from within and compressed from without. There were sometimes most agonising cramps in the legs.

The disease was most treacherous and fatal when the symptoms

¹ *I. M. J.*, December 1836, p. 623. Dr. R. Brown has also described this outbreak, *Ibid.*, May 1, 1838, p. 261.

did not run high. When the patient had not been in his usual health in barracks for a week or ten days, he reported himself ill, but could not tell what was the matter. He appeared unable or unwilling to answer questions. He had no pain or sickness; he only complained that he could not sleep or eat. For several mornings after admission, he would answer, 'Very well, sir.' Pulse, skin, and tongue healthy. When, however, he was desired to walk, he could not stand, or staggered like a drunken man. He had lost command of his limbs, and nervous energy seemed to have left him.

Subsequently, perhaps, he would have evening exacerbations, somnolency, immobility of pupils, or bilious vomiting, or watery purging two or three days before death. Others lingered and often sank gradually without these symptoms. One of the most usual characteristics of this disease was debility, moral and physical. This was great and out of all proportion to the previous febrile excitement. A patient, after one, two, or three paroxysms of fever of moderate severity, remained for weeks and months in a state of debility, having perhaps an occasional exacerbation of an afternoon, or not. In some cases of this kind, the patient walked about, recovered appetite, and had a clean tongue and regular bowels. If discharged to duty, however, he soon had either a relapse¹ or an attack of some other disease. He could not recover his lost strength.

Extreme apprehension of a fatal termination was frequently observed, but not in the most serious cases. The men seemed determined to die—to lose all confidence in medicine. This condition was indicated by the manner, countenance, and tone of voice. When this feeling was unaccompanied by other symptoms, very little danger was to be apprehended. It will be borne in mind that despondency was a marked feature in the outbreak in the previous year, described by Mr. Raleigh.

The most alarming feature was collapse, 'which came across the hospital like a blight, prevailed for three or four days, and then disappeared. On these occasions, the weather was generally cloudy, rainy, and thundering. This exhaustion of the vital powers seized some men suddenly, who had been in hospital several days for other diseases. It often appeared in subjects convalescent from slight attacks of fever. It often happened that the majority of the patients in hospital had more or less sinking of the vital powers. The thready or imperceptible pulse, the drenched skin, and the prostration of strength reminded one of cholera.'

'The patient did not appear sensible of his awful situation. His tongue and breath were not cold; neither had he cholera evacuations. No patient was brought to hospital in a state of collapse, with the

¹ In describing the malarious pernicious fevers of the Havana, Dr. Sullivan states that a tendency to relapse or recur is one of their marked characteristics.

exception of one who, also having congee-coloured' [rice-water] 'evacuations upwards and downwards, was admitted under the head of cholera.'

'When irritability of stomach and collapse co-existed, the case was hopeless. It was observed as frequently in intermittents as remittents; was not preceded or accompanied by horripilation or shaking nor was it consequent upon a febrile exacerbation. With this sudden sinking, there sometimes existed internal heat and thirst. The extremities were often deadly cold, while the trunk was warm. Patients did not complain of cold; they seemed to be insensible of the great loss of animal temperature.

'Some remained in collapse from nine to twelve hours. Recovered by stimulants, they had another attack, and got well again.' 'Could this state of collapse,' the able medical officer inquired, 'be the cold stage of the paroxysm of a highly congestive form' [of fever], 'or is it not more probable that it was some other disease to which the patient was predisposed by fever?'

Tonics, wine, and beer were administered in vain. The collapse, it is urged, was not the effect of treatment, because it occurred in men who had been neither leeches nor bled. Dr. Jackson had no doubt that an ordinary blood-letting would have induced or aggravated this condition. In some cases, severe from the first, there supervened nocturnal perspirations, so profuse as to drench the bedding. This did not relieve, but debilitated the patients, who sank under the disease. There were, in four months, 232 cases of fever, of which 100 were serious, with 22 deaths.

There must have been something very unusual in the zymotic condition of the atmosphere at this time. The pernicious remittent fever in Bengal, described by Mr. Raleigh, occurred in 1834; the pernicious fever of Deesa was observed twelve months later; Pali plague broke out in 1836; and this latter disease, an unusually severe endemic of mahamurree in Gurhwal, and relapsing fever in Bareilly, Rohilkund, and the districts around Delhi, were contemporaneous.

I shall have to speak again of this Deesa fever and of other similar outbreaks in India under the head of cholera, when showing that algide pernicious remittent fever and Asiatic cholera are closely linked together.

The above history of the Deesa outbreak illustrates a very noteworthy fact, frequently observed in India, viz., that both European and native troops are particularly liable to fever and other disease in the *second* or *third* year of their occupation of a malarious district. The first year this regiment was at Deesa, they had no sickness, with the exception of a few cases of heat-stroke and bilious remittent fever. As the narrative cited above shows, the second year brought with it

a heavy sick-list and many casualties. So also Dr. Finch proved,¹ by ample statistical data, that, to up-country sepoys 'the third year is pre-eminently the fatal year of their residence in the malarious climate of Lower Bengal.'

Terai Fever.—Cases of this deadly pernicious remittent have generally been seen at hill stations, and in the plains immediately below them, in persons who have become fever-stricken while passing through the jungly *terai*—that water-logged belt of soil, overcharged with decomposing vegetable matter, which renders the foot of every hill in every country more or less insalubrious; but which, as it is seen stretching in miles of the wildest and rankest vegetation at the foot of the Himalayas, engenders malarious poison in its most concentrated virulence. It is considered that the *terai* is most insalubrious from the middle of March until the middle of November. Residents in the hill stations are also liable to attacks, especially those whom sport, occupation, or duty lead downwards within reach of the poison.

Having written very fully upon the habits of Indian malaria in my work on 'The Means of Preserving the Health of European Soldiers in India,' I say little in these pages on that important point. I may merely state here that all authorities have held that it is especially dangerous to *sleep* while crossing the *terai*; and I may mention, as a suggestive hint to the investigators of the habits of the *Bacillus malarie*, that Kenneth Mackinnon says that, where a person is exposed to high malarious influence, 'the use of a gauze veil or mosquito curtains is worthy of notice.'

Before quinine came into use, and early in our occupation of the hill stations, when the roads through the *terai* were bad and narrow, *terai* fever was very prevalent and deadly. Allan Webb² says that it killed the very letter-carriers, in their passage through the jungle at the foot of the hills. And Macgregor wrote that individuals seized with fever in the *terai* below Almorah seldom or never recovered.

The fever of Indian *terais* and hill jungles of the greatest density is now remittent of the most aggravated intensity, with a strong tendency to various pernicious complications. Its type has varied at different times and in different localities.

Those who observed the disease soon after our occupation of the hill stations agree in describing it as remittent fever. Writing in 1843, Dr. Macdonald described jungle or *terai* fever as 'bilious remittent.' Dr. Kenneth Mackinnon (1847) classed it with the 'paroxysmal remittent.' No Indian fever displays so marked a tendency to deadly pernicious complication as this does. In treating of *terai* fever (as he saw it, probably *cir.* 1855), Mr. Hare writes that, as an instance, he

¹ *I. M. J.*, 1844, p. 160, on 'The Effect of Change of Climate on the Health of the Native Army.'

² *Pathologia Indica*, p. *213.

may mention the Residency Surgeon of Nepaul. He passed through the *terai* incautiously, and died on his arrival at Benares, in a state of blue collapse very similar to cholera. Mr. Hare describes, as the worst case he ever saw, that of Assistant-Surgeon W., a strong, active man, and celebrated for the fatigue he could undergo in shooting.¹ Dr. W. was delayed for one night in the *terai* at the foot of the Nynee Tal Hills; and, on his arrival at Simla, had all the symptoms of delirium tremens, so much so, that many thought, though he was a strict water-drinker, that he was suffering from intemperance. This strong, healthy young man, from a few hours' exposure, became blue and livid; he staggered when he attempted to stand, his hand and tongue were tremulous, his eye dull, and, though he moved about, he seemed unconscious of what he was doing, and he died, after some days, with coma.

Kenneth Mackinnon, writing some years before this case occurred, cites Watson's Lectures to the following effect:—'You will sometimes find a state resembling delirium tremens left after subsidence of acute inflammation of the parts within the cranium, and requiring the treatment of delirium tremens.' Mackinnon says: 'Dr. Watson might, I think, have added, this state is particularly apt to affect those addicted to the use of spirits.' Dr. Morehead gives a case (20, page 79) of this description; but, doubtless, there was no such causation in the case of Dr. W., a man thoroughly well known and much valued in the service. The pernicious character of his case was evidently attributable to exhausted innervation, due to the presence in the system of an overwhelming dose of malarious poison.

Dr. John Sullivan, whose admirably graphic description of pernicious fever in Havana is cited by Aitken,² would class the case of the Residency Surgeon of Nepaul as *febris p. algida*; that of Dr. W. appears to have partaken of the characters of both *febris p. algida* and *febris p. comatosa*, in both of which 'malaria directly attacks nerve-power.' There can be no question that the great fatigue and exposure of the journey often precipitates exhaustion. This doubtless occurred in the terribly rapid case of an esteemed lady, whose forced journey, straight through from Darjeeling to Calcutta, would have greatly tried a robust constitution. As I have already mentioned, I saw Dr. W. in Calcutta (it was before the railway extended to Benares) a short time before his death. He must, therefore, have had a most trying palkee

¹ I saw Dr. W. shortly before he left Calcutta on his fatal journey. He appeared to be a very healthy man; but it is noteworthy that Indian sportsmen, however vigorous they may be in appearance, not unfrequently succumb to attacks of fever and of other diseases—snipe-shooting and tiger-killing in swamps and jungles having implanted malarious influence in their systems, thus impairing their power of resisting and sustaining the attacks of acute disease.

² Vol. i. p. 438.

journey. Upon this point Dr. Sullivan says that, 'like other pernicious fevers, the comatose form especially attacks those who, after a long residence in a marshy district, remove into another free from malaria.' So Macgregor observed in India, more than forty years ago, that, 'provided the traveller does not expose himself, when in the *terai*, to the direct rays of the sun, the miasma does not immediately produce its bad effects; but, after the person reaches Bareilly, Moradabad, or other stations in Rohilkund, he is seized with jungle fever, and quickly falls a victim to it.' In like manner, Sullivan writes of *comatose pernicious fever*. 'Insolation is occasionally a most powerful cause. A man who leaves a marshy district and travels a long way, exposed to a burning sun, is very subject to an attack of malarious apoplexy.' My late honoured friend, Sir John Forsyth, told me that officers travelling from the Nerbudda Valley across the hills to Bombay, used to be struck down by this deadly fever with the same pernicious complications.

Dr. Sullivan found, in comatose pernicious fever, no marked distinctive brain lesions. The pia mater was somewhat injected, the cerebral substance redder than usual, and there was some serum in the ventricles.

Allan Webb describes,¹ under the designation of 'Hill Colic,' what was, in 1840, 'a very fatal and also a very common disease at Simla.' This was clearly *algide pernicious fever*. Natives were the subjects of this malady. The seizure was always sudden, and characterised by great pain in the bowels, which caused the patient to roll on the ground, and to keep the hands firmly pressed upon the abdomen. The expression of face was anxious and peculiar, the eyes sunk, the skin cold or cool, the tongue cool, the urine arrested, no stools. The symptoms of congestion increased; pulse was lost, the belly became hard, tender, and swollen. The patient's shrieks were converted into low moans. No blood could be obtained by venesection, and the patient died; the mind and muscular powers keeping their force unimpaired to the last. Sometimes there was vomiting, and occasionally spasms, as in cholera; perspiration rarely occurred till the last, and then it was cold. Dr. Webb says that the post-mortem appearances were not uniform, but that the following were most characteristic:—Head and chest healthy. Mucous membrane of the stomach dotted all over with sooty-looking black points and streaks, and coated with thick ropy mucus. Duodenum presented patches of ecchymosis. Mesentery redder than natural; a vivid red line at its junction with the intestine; ileum congested, of a dark red colour; mucous lining softened; no lymph effused; upper part filled more or less with black sooty secretion, without a trace of bile, perfectly inodorous, and very like the black vomit of yellow fever.

¹ C. M. T., vol. viii. part 2, p. 296, 1842.

There was less of this in the lower portion. The outside of the cæcum had a puckered-up appearance ; its mucous surface was slightly eroded. The bladder contained a small quantity of dark-coloured urine. Spleen enlarged ; liver congested ; abdominal veins distended with dark tarry blood, without any attempt at coagulation. The general appearance was that of robust health ; muscles finely developed. The prognosis was favourable when reaction was fully and early set up, if stools could be procured, and the secretion of urine restored. When, however, reaction was long in setting in, '*it was followed by severe fever, with determination to the head,*¹ and to the mucous surfaces of the bowels, &c.' If seen early, the disease might terminate favourably in twenty-four hours ; when prolonged beyond this time without relief, it was generally fatal. Even if the patient did recover, the subsequent debility was great, and the convalescence was protracted.

Mr. Hare writes :²—' In what is called hæmorrhagic dysentery, described well by Dr. Raleigh in Calcutta, and of which I had numerous cases in Burmah, during the extreme congestion of the portal system in this algide fever (for, in reality, it is that disease, and not dysentery) the blood at length oozes from the gorged mesentery, through the coats of the bowel, and the patient dies, passing pints of pure dark blood which will not coagulate, and his skin is like ice to the touch.' Mr. Hare gives the case of an officer serving in Burmah, who, being known to dread the climate and its discomforts and, being a stout, tall athletic young man, was supposed to have nothing the matter with him, and was suspected of wishing to get away from his regiment. He was found lying in bed, quite sensible, but his face and lips were blue, his breath was cool, and his hand felt like a dead man's. He lingered for some days in the same cold state ; at last, the blood found vent from the bowels, and he died with profuse hæmorrhage. In such cases, there is no general ulceration of the intestine, but a general oozing of blood from the mucous surface throughout to the anus.

Dr. Sullivan insists that 'between pernicious choleraic fever and Asiatic cholera it is almost impossible to diagnose.' A middle-aged Mohammedan, very well set and muscular, but of worn appearance, was brought to my jail hospital at Chittagong wildly maniacal. For many hours, his fury and strength were perfectly astonishing. Until he could be put under restraint, his chief efforts were to escape, scrambling up walls, and tearing heavy doors from their hinges. He became suddenly collapsed, as if exhausted by his rage. He appeared to be in the algide stage of cholera, *except that there was neither purging nor vomiting*. He sank in a few hours. The only remarkable post-mortem appearance was that the mucous surface of the small intestine, otherwise empty, was marked throughout its course by a

¹ The italics are mine.—N. C.

² *Loc. cit.* p. 42.

defined streak of thick, greenish-black, tarry fluid, about an inch and a half wide.

This case, much as it resembled one of algide cholera, appears to have resulted from shock produced by an overwhelming dose of fever-poison operating at a moment when the nervous system was prostrate from inanition and fatigue. I feel very little doubt that, had I been able to examine the poor fellow more carefully than I could during the stage of frenzied excitement, I should have found that he was in strong fever.

We are told by William Fergusson that it often happened to a well-seasoned soldier, mounting the night-guard in perfect health, to be seized with furious delirium while standing sentry; and, when carried back to his barracks on Monk's Hill, to expire in all the horrors of the black vomit.

I think it clear that Dr. Webb's cases of algide pernicious fever and this case of the Mohammedan are links in a chain which connects the disease that must next engage our attention—*choleraic pernicious fever*—with true algide cholera. Dr. Geddes made early mention of this type of pernicious fever.¹ He observes: 'In some instances' [of fever] 'the evacuations have presented the characteristic appearance of the egesta of epidemic cholera. In several of these cases the countenance, too, has become shrunk, and the patient has assumed somewhat of the peculiar features of this disease.' I had one solitary distinct case of this kind many years ago in my cholera ward in Calcutta. The man had strong fever, with rice-water cholera stools. I pointed it out to the students as a case nearly resembling those of pernicious fever described by American writers. I more frequently met with cholera-like cases in which a short, but very marked stage of collapse was suddenly followed by strong reactional fever. I now seem to see the livid, sunken countenance of a handsome young European, as he was brought through the door of my ward to a bed, where I was standing ready to receive him. He passed a rice-water stool, the characteristic appearance of which I well recollect, because its odour was so peculiarly offensive that I felt much nauseated when I examined it. His extremities were cold when he came in, but was in strong fever before I left the ward. I heard, three years ago, of a case of this kind in a young gentleman who had recently died, as it was considered, of cholera, in Calcutta. I need scarcely say that, in ordinary cases of Asiatic cholera, rice-water stools occur only during the stage of collapse.

Sir Joseph Fayrer says: 'An attack, beginning with symptoms of fever, may end with cholera, or *vice versâ*. And even recently, such an epidemic was devastating Amritsur in the Punjab.' He cites Mr. Anodochurn Kastogiri, a learned Bengali physician and graduate of

¹ *Clinical Researches into the Diseases of India*, p. 123.

Calcutta, who says that ancient Hindoo authors mention symptoms of cholera as being prominent in a certain type of fever called *jewarutishar*, literally, fever with excessive diarrhoea.

There are many Indian localities—such as the forest jungles of Chittagong, which were considered as imperilling the life of any European who ventured into them—where, although the malarial poison does not appear to be so extremely concentrated and deadly as that of the great Himalayan *terai*, it is very dangerous. Remittent fever, far more ‘*pucca*,’ i.e. severe and obstinate, than ordinary remittent, but not usually characterised by pernicious tendency, frequently attacks surveying officers, elephant-catchers, and hunters. I have seen a good deal of this type of fever, in both its ardent and congestive forms; there being severe head complication in the former, and troublesome abdominal congestion, especially hepatic, in the latter. Taken early, this remittent is usually amenable to quinine.¹

In the present day we hear far less of pernicious *terai* fever than we formerly did. Travellers are prepared with quinine against it; the roads are more open and better; transit is quicker; night journeying at the worst season is avoided. Large tracts of *terai* are now under tea cultivation; the European planters being liable to intermittent fever. It may be that the type of disease has become mitigated; still, as long as virgin forest remains, men will venture into it, and get remittent fever of grave character, which Indian physicians must be prepared to treat with a full knowledge of the history of *terai* fever in its deadliest forms.²

TREATMENT OF THE MALARIAL REMITTENT OF THE PLAINS.

Only a short time before I went to India, the antiphlogistic system, of which bleeding and mercury were the chief arms, was the ‘sheet anchor’ in the treatment of remittent fever. I know men who are still healthy who were bled and salivated for malarial fever, but who probably would have been much healthier, and who would doubtless have suffered less at the hands of dentists, if their cure had been

¹ My late friend Dr. Allan Webb described a fever, of which he saw cases at Simla in 1840, as a ‘modified’ ‘malignant’ typhus. Perusal of his account of this disease (*C. M. T.*, vol. viii. part 2, p. 277, and *Pathologia Indica*, p. *213) shows that these were probably cases of mahamurree, as, ‘in the worst cases brought to hospital, buboes were met with both in the groin and axilla.’ Webb considered it likely that the two fevers were identical.

² Drs. Campbell and Mackinnon speak of the Mechis, an aboriginal Mongolian flesh-eating race, who cultivate small holdings in the Terai, who live in perfect security in parts of the forest which, at some seasons, it would be certain death for any other person to visit. The Dangurs, of the Chota Nagpore Hills, also a strong aboriginal race, appear to enjoy a like immunity.

effected by quinine. I, ill directed by my books of reference, treated fever with calomel and VS. in my first voyage to India, in medical charge of recruits, in 1848. Full and interesting histories of this heroic and spoliative treatment in India have been written by able authors.¹ It prevailed for many years; writers of authority who zealously advocated it are now living. It vitiates the practical utility of the otherwise still invaluable works of men who used to be the leaders of medical opinion in the East—Twining, James Johnson, Kenneth Mackinnon, and Ranald Martin. A general idea of the prevailing opinion, to deny which was ignorance and heresy, may be formed by reading the following passage from the chapter on remittent fever in a sensible book on the principal diseases of India, published in 1843, by Dr. W. L. Macgregor: 'If the lancet and calomel fail in arresting the progress of the disease, the result is generally fatal.' To many officers of experience now practising in India this mode of treating malarious fevers must appear irrational and unjustifiable, but it was perfectly in accordance with the opinions acted upon by the profession at home. It is almost needless to say that, until VS. went out in India, it was practised everywhere in nearly all inflammatory diseases and fevers. Even at the great London hospitals, people used to visit the surgery to be bled by the dressers every 'spring and fall,' and whenever their own sensations or the opinion of their friends that they were too 'full-blooded' appeared to indicate this measure; while the cuppers exercised their own discretion in performing the barbarous operation of C.C. upon all who demanded it.²

Further, Indian physicians employed bleeding in fevers upon a *rationale* which, however incorrect, was at least perfectly clear and definite. They held that it was needful to prevent and to subdue 'ardency,' and to avert and to relieve *abdominal congestion*. Again, mercury would not act upon the system until the force of the circulation had been reduced by blood-letting, and blisters could not be employed if not preceded by active depletion.

Many of our early authorities on the treatment of Indian fevers were 'Company's' medical officers whose practice lay chiefly among European troops. Their patients were generally over-fed and lazy, drank spirits freely, and went about recklessly in the sun; consequently, 'ardency' was a marked feature in the fevers of the hot

¹ By Edward Hare, 'Malarious Fever,' *I. A.*, No. xviii. p. 1; and by Joseph Ewart, *Ibid.*, No. xiv. p. 1, 'A Review of the Treatment of Tropical Diseases—Malarious Fever.'

² I had just written this when a valued contemporary, who studied in Edinburgh, looked in. I read the passage to him. 'Yes,' he said; 'and we used to cup one another. I have still the marks on my arm; Mac—— cupped me, and I cupped him.'

weather. It is probable that, in Twining's time, the type of intermittents and remittents displayed more ardency than it does at present. Very possibly these 'ardent fevers' then demanded bleeding and bore it well, as London typhus did sixty years ago. The worst complications of these fevers, cerebral and abdominal congestions, were then considered, perhaps justly, to be essentially inflammatory. I do not believe that, in my time, they were inflammatory at all; if they were, quinine commanded them. As I have already remarked, there was not much of this extreme acute visceral congestion in the Bengal remittents of my day, except where the use of quinine and other treatment had been neglected until the patient was moribund or nearly so. Hare is certainly right in saying that quinine prevents congestion in these fevers. Where we had opportunity of treating our cases early in Bengal, we were so little apprehensive of serious internal complications that an able contemporary of my own speaks of 'that bugbear, abdominal congestion.' But there cannot be a doubt that, whether it depended upon the type of the disease or the non-administration of quinine, grave abdominal congestion was of frequent occurrence in Twining's day; and, even in my time, severe splenic lesion was characteristic of the first attack of Mutlah and Peshawar fever. If the type of extreme ardency should ever again characterise the fever of Europeans in the hot weather, it is not impossible that it would demand loss of blood; but, if it could fall to my lot to encounter the outbreak of such a fever, I would certainly begin with the use of quinine, guarded by ipecacuanha, in the belief and full hope that I should succeed in quelling the ardency, without the use of even a single leech.

In 1848, Kenneth Mackinnon regarded mercury in fevers 'as a specific to check the consequence of inflammation.' It may be asked, How came it that, for many years, highly educated physicians, observant and deep-reasoning men, went on salivating the unhappy victims of remittent fever? The answer is plain. Mercury is quite as potent as quinine is in 'curing' a paludal fever which immediately threatens life.

Kenneth Mackinnon saw this truth darkly. He says (p. 227): 'I believe, though I am not quite sure, that it is a febrifuge.' Edward Hare, who overthrew the practice of salivation in Indian fevers, perceived it clearly. He taught¹ that mercury is an antidote in malarious fevers. But how evil an antidote! How contrary was its use to the wise injunction of Henry Marshall Hughes: '*Combat disease by those means which are least expensive to the patient's constitution.*' Treated by calomel and bleeding (calomel required bleeding because, until the ardency of fever was subdued by VS., mercury would not salivate), the

¹ 'Malarious Fever,' *I. A.*, No. xviii, 1865.

victim of an Indian paludal remittent escaped with bare life and a ruined constitution.¹

We object to the mercurial treatment of Indian fevers as we do to variolous inoculation, not because it is inefficient, but because we have, in quinine as in vaccination, a remedy which is not only valid, but perfectly safe.

The fight of advancing science and good sense against the mercurial treatment of Indian fevers was prolonged and fierce. There now lies before me a very rare little book which relates an early and most singular episode in this contest. It is known as 'Halliday's Memorial.' Dr. A. Halliday, a Presidency surgeon in Calcutta of fifteen years' Indian service, a clever man, but rather ardent controversialist, was, in the year 1821, suspended by Government from employment and from the Service for arguing rather too warmly to the Medical Board, one of whose members was a strong believer in calomel, that this drug was used with dangerous recklessness at the General Hospital. Four cases which he cited will satisfy the reader of the present day. V. got 686 grains of calomel for an attack of fever. J. got 864 grains for the same disease. For dysentery, L. got 695 grains, and B. 974. It need scarcely be added that these patients died.² I am not an advocate for the utter abandonment of the employment of calomel in deadly Indian remittent, but I used it only in cases where life was evidently threatened.

In regard to bleeding. For many years before I left India, VS. had been practically abandoned. During the first six years of my Indian practice, I always carried a case of lancets. I never used these but twice, and then in vain—once in a case of apoplexy, on another occasion in one of injury to the head. In old times, my hospital used to possess a large stoppered bottle filled with bleeding-lancets. About thirteen years ago, having a troublesome boil which I carefully concealed from my colleagues, who were addicted to the use of the scalpel, I sent to the hospital for a bleeding-lancet, and could not get one! I was more fortunate at a private druggist's. Still, I did not consider it needful to send in an emergent indent, upon the Apothecary-General,

¹ I am quite willing to admit that we, of the present day, who are anti-mercurialists, have resigned a power which the greatest of our predecessors exercised with large success. I say 'the greatest' because I have not the slightest doubt that men of the highest discrimination and judgment, like Abraham Colles, possessed a most valuable remedial agent in mercury. A few years ago a careful perusal of Colles's work on Syphilis and the Use of Mercury convinced me that, *in his hands*, the drug was both safe and efficient. I happened to be case-taking in a ward at Guy's Hospital when Bransby Cooper and Colles came alone to a woman in whom nearly everything had been tried in vain for the cure of an ununited fracture of the humerus. 'If I had that case,' said the Dublin surgeon, 'I would put up the arm in splints and touch her gums.' This was done, and the bone reunited as if it had been recently broken.

² Pp. 174-75.

for bleeding-lancets. I have discussed the question of bleeding in Indian fevers with senior medical brethren who had practised VS. and undergone it in their own persons. The arguments which recommended it were clear. It abated 'ardency'; it cooled the skin and lowered the pulse; it enabled mercury to salivate; it relieved headache, splenic distension, and the pain of pleurisy and hepatitis; it freed the breathing in pneumonia. Thus it satisfied the physician and the patient, and also the bystanders, who, in India, have always been energetic in demanding that 'decisive measures shall be promptly adopted.' A distinct impression upon the disease, apparent to everyone, was produced: thus every bleeding was regarded by all as a triumph of science over disease—quite irrespective of the final result. I know the record of a case illustrative of the use of VS. by an Indian physician, whose memory I esteem and honour so much that I will not name him, which, but for its sadness, might now cause a smile as a piece of grim humour. The narrator met a brother officer at a consultation in Calcutta. As they were separating, the colleague spoke of a pain in the right loin, which had troubled him for a day or two. This, on examination, was found to be due to inflammation of the posterior part of the right lobe of the liver. The narrator adds: '*Although* he was bled three times, he had a difficult recovery'!¹

When I first became secretary to the Calcutta Medical Board, in 1855, fever was very prevalent in Peshawar, and was generally attended with splenic enlargement. It was the fashion to apply multitudes of leeches over Peshawar spleens. Leeches were so costly that, in one instance, the Board felt justified in questioning the expenditure. The very able surgeon declared that he had prescribed, and even personally supervised, the application of every leech. A member of the Board, who was a good arithmetician, went closely into the question, and gave us several very striking calculations, one of which showed that the bulk of leeches reported to have been consumed, in one month, in one regimental hospital, would, in the aggregate, fill an ordinary coffin! Nearly twenty years after this happened, I was told by my house-physician, at my morning visit, that the next patient, just admitted, complained of hepatic pain. Without speaking to the man, I uncovered his abdomen, and then ventured confidently upon the questions—'In what regiment did you serve at Peshawar twenty years ago, and when did you take your discharge?' The man appeared utterly astonished and exclaimed: 'Did you ever see me before? How came you to know that?' 'No,' I replied; 'you are quite a stranger to me, but we doctors know many things "not dreamt of in your philosophy."' The explanation is easy. This was a middle-aged man, with some traces of soldierly bearing, much anæmiated. A single glance at the left hypochondrium showed an enormous constel-

¹ The italics are mine.—N. C.

lation of leech-bites; and my knowledge of Indian medical practice, during the previous quarter of a century, told me that it was only in the regimental hospitals at Peshawar that leeches had been applied over big spleens in such profusion within that term.

This heroic mode of treating enlarged spleen did not, however, at all originate with Queen's surgeons at Peshawar. After recommending VS. guardedly in inflammatory remittent, Mackinnon (*op. cit.* p. 239) says: 'Leeches are always advisable, even when the complications are moderate in degree, and particularly when the exacerbations and remissions are well marked, . . . for this shows us more than anything that we have to deal with a marsh fever, the issue of which is always more or less uncertain.' As the MS. of this chapter was lying on my table, a brother officer told me that, having been attacked with malarial fever in the jungly Soonderbuns, on his way to Chittagong, and being taken into that station insensible, the surgeon bled, leeches, and blistered him within the first four-and-twenty hours. Placing his hand over his left hypochondriac region, he added: 'I have still scars of thirty-eight leech-bites here.' The types of fever were such at Chittagong, when I joined several years later (in 1849), as certainly would not have admitted of such heroic depletion. In his long and very distinguished subsequent Indian career, Dr. —, although strong and active, always appeared anæmic. After many years' retirement, he is florid and enjoys a good measure of health.

I have often thought that some laborious medical book-worm might do well to compile a list of *maxima* and *minima*, giving, for example, among a thousand other extreme facts, the largest number of leeches applied, the largest quantity of blood drawn by VS., and the greatest weight of calomel swallowed, in the cases of patients *who survived*. The following example¹ would almost be worthy of a place in such a compilation, as showing how much depletion was formerly employed and recovered from—(did the constitution of this patient ever fully recover?)—in India. The italics are mine.

A young European gentleman, aged twenty, was attacked with remittent fever. He was found to be labouring 'under a rather acute paroxysm; the tongue being much furred, the pulse rather quick and full, with pain somewhat severe in the temples and back of the head. *A considerable quantity of leeches were applied to the temples*, and calomel and antimony with purgatives were administered. Next morning, there was apyrexia or decided remission, but secretions were deficient, and the tongue was still coated in the centre. *A similar treatment was pursued*, the paroxysms returning about noon and continuing till morning, increasing in severity. The pain at the occiput was particularly marked. It would be uninteresting to detail the daily treatment; suffice it to say, *it was antiphlogistic*. He was

¹ Abridged from the *I. M. J.*, New Series, vol. ii. for 1837 p. 276.

bled to syncope, had leeches repeated, a scruple dose of calomel, with smaller and repeated doses at intervals, followed in four hours by compound infusion of senna and sulphate of magnesia. On the fifth day, he was seized with a fit of epilepsy.' His medical attendant attributed this mainly to a fall from his horse six weeks previously, when he pitched on the back of his head, and suffered from moderate symptoms of concussion. 'During and subsequently to the fit, the pupils were enormously dilated' [From head mischief, or from loss of blood?—N. C.], 'the febrile excitement increased, and the pain in the occipital region was severe. *Fifty leeches were again applied*' [the word 'again' appears to signify that fifty were applied on each of the three previous occasions]; 'a large blister to the back of the neck, and a scruple dose of calomel followed by compound powder of jalap and scammony. Three hours afterwards, and before the remedies had been administered, another epileptic paroxysm occurred, the convulsions of the extremities being severe, and the patient remaining sensible during the fit. He was seen immediately on its subsidence. *He was again bled freely from the arm*; the last mentioned' [blister and drugs] 'which had arrived were simultaneously administered. The pain of the head greatly subsided.' The bowels acted freely, the tongue became clean, 'but the pulse was still somewhat sharp' [Shall we say hæmorrhagic?]. The skin acted freely. He was kept under the action of pills composed of a quarter of a grain of tartarised antimony, six grains of calomel, and six of compound extract of colocynth, taken every two hours. 'From this period, the more alarming features of the disorder ceased to manifest themselves. His mouth became slightly affected by the calomel; two or three slight paroxysms of fever occurred, but it assumed quite an intermittent form, which yielded to exhibition of sulphate of quinine every four hours, and attention to the bowels. He has now recovered.'

We have seen that, when Kenneth Mackinnon wrote in 1848, he admitted the use of bleeding and calomel in Indian fevers, but the reader who will peruse his fourth chapter will perceive that his admirably careful and thoughtful mind had begun, at least, to see that a great change of opinion and practice, the light of which had already dawned upon him, was at hand; he consequently surrounds his heroic spoliative treatment with injunctions of moderation and caution in its employment. Everywhere, at that time, VS. was beginning to go out of use, and quinine was gaining accessions of reputation. Long previous to this, Mackinnon had reservedly acknowledged the potency of quinine in Indian fever. In 1838, he wrote: 'I cannot say that I have ever seen the sulphate of quinine of much use given with a hot skin, but in the marsh fever, when a paroxysm is ended, it is our sheet-anchor, and he who waits for a clean tongue

on which to give it is, in my opinion, risking the life of his patient.' ¹ In 1847, the year preceding that in which I went to India, Mr. Edward Hare came boldly forward with that which was considered at the time as a grand heresy, but which was recognised by many as a great truth, and which, with considerable modification, was soon adopted as our established practice. Citing the authority of American physicians, Mr. Hare boldly declared that 'Quinine may be given in the largest doses, whether there are head symptoms, delirium, coma, or pain in the liver. Whether it be in the hot stage or the cold, quinine is not only safe in all forms of malarious fever, but its certain cure. And in cases where there is danger to life, the earlier and the larger the doses of quinine which can be given to the patient, the better.' Mackinnon received this bold opinion with great reserve. I italicise the words which convey the main objection which he and many others made to the new idea. '*In fever we have been watching, and in remittent with the feeling that life or death hung upon it, for the time when we could begin quinine.*' Now we are informed that waiting is in every point of view loss of time; that quinine may be given safely in the very climax of fever; that it will prevent the congestions and complications which end in death.' This waiting for remission and complete intermission was the great error of old practice over which Hare triumphed. And Mackinnon's last review of the treatment of remittent, published in 1855,² shows that his large and candid mind had then almost adopted the truth of the new doctrine in its entirety, although he had not abandoned the opinion that, especially in the continued fevers, the employment of 'mild depletion' is 'useful and advisable.'³ So strongly, however, was the deadly error of waiting for full remission rooted in India that, up to the day of my retirement, ten years ago, it was occasionally cast in my face. I would inquire, on being called to consult upon a case of remittent of grave type, 'How long has he been ill?' 'Eight days.'

¹ Foot-note, p. 207.

² *I. A.*, No. v. p. 154.

³ At first, people were severe upon Hare for saying that, from James Johnson's time until 1847, the only advice had been to bleed and give calomel purgatives, but to be very careful not to give quinine too soon. This statement was shown by my friend, Dr. John Macpherson (*Bengal Dysentery, &c.*, with a Notice of the Use of Quinine in Intermittent Fever, 1850), to have been rather too sweeping. Although Hare gave due credit to others, Dr. Macpherson's history of the introduction into practice of the free use of quinine, early in the progress of remittent fever, is of great value. Hare's way had been paved by Maillot in Africa, Hille in Surinam, Byrne and other American physicians, Geddes, Wright, Cadenhead, and Eyre in Madras, Morehead in Bombay, and by Corbyn, Twining, J. Murray, John Macpherson, and Ranald Martin in the Bengal Presidency. Nevertheless, although Hare was not altogether a discoverer, he was a great reformer, and there can be no question that, almost from the day on which he placed his views before the profession, in November 1847, his energetic advocacy of quinine put an end to the evil reign of bleeding and calomel in Bengal.

'In what doses are you giving quinine?' 'He has not had any. I have endeavoured, but in vain, to obtain sufficiently marked remission.' 'Yes,' I would add, 'he will probably die in a few hours, despite any treatment which you can now adopt.' Everyone, I think, now agrees that (except where death is threatened in the cold stage, or where the hot stage is so violent as to assume the heat-stroke character) it is better not to give quinine in intermittent until the very first commencement of the sweating stage. In a common intermittent, a large dose of quinine, taken a little before or in the hot stage, generally gives a headache which is so severe as to be worse than the disease itself. I have repeatedly watched the fierce conflict between the fever and an heroic dose of its antidote. Its intensity is such as to recall Crashaw's lines—

'Go, now, and with some daring drug
Bait thy disease; and, whilst they tug,
Thou, to maintain their precious strife,
Spend the dear treasures of thy life.'

Consequently, although I fully agreed with Hare in his main principle, I did not give quinine *in large doses* from the first in all stages of malarious remittent. In a severe case, I preferred to give two grains of quinine every hour throughout the height of the disease, guarding each dose with a sixth or an eighth of a grain of ipecacuanha when there was delirium or coma. This practice was attended with so much success that I pursued it undoubtingly until I left India.

When I first published these observations, my rather incautious use of the expression that paludal remittent is 'curable by quinine,' in the chapter on true enteric fever, *M. T.*, page 364, of September 27, 1879, brought upon me the inquiry, 'Have you always been able to cut short a case of severe remittent by quinine?' This question was, I think, sufficiently dealt with previously in my definition of the QUININE TEST of Indian fevers—page 131, of August 2, 1879, and page 83 of this volume—in which I say that neglected and moribund cases are often found to be beyond the power of quinine. My mind travels over a multitude of native cases who were admitted to hospital in an absolutely hopeless or positively dying state—cases brought, with great fatigue and exposure and want of supporting diet, from remote country places, after the disease had gone on unchecked for days; and I recollect many other cases of slow recovery due to the same causes. But, in Europeans, who generally seek aid at once, I had a large amount of good success. In uncomplicated cases of remittent, taken early, the QUININE TEST will rarely fail; and I believe that, *if recovery be possible, remittent fever will always yield, albeit often very slowly, to quinine*, backed by due support and careful nursing.

When I believed that quinine would suffice, I relied solely upon it, but, when I saw that a patient was threatened with death, I endeavoured to get his system under the influence of the other great antidote, mercury. I did this as early as possible, i.e. immediately I was called to the dying man (not discontinuing quinine), but never with perceptible good result in cases of ordinary remittent, probably because the specific was employed too late. It goes very hard with the subject of remittent fever when his case has advanced so far as to resist the commanding power of quinine. In the cases where convulsion takes the place of rigor in the cold stage (where I consider that the type is usually pernicious intermittent—not remittent), my experience leads me to place a high value upon one or two doses of calomel in combination with quinine, given promptly. Steady nourishment, day and night, *always by night*—in severe cases by the rectum—is indispensable in remittents. At the outset, the bowels must be thoroughly cleared by aperients, but to cause intestinal irritation by drastics is, in the last degree, objectionable. In natives, a minor but important precaution must never be neglected in cases of fever and in other severe diseases. When brought to us, especially in the cold season, their skins are frequently so perfectly unclean and dry, and often so beset with aggravated chronic scabies, that we cannot hope to treat fever or to employ diaphoretics with any chance of success until the *exuviae* are removed. With this view it is safest, to avoid chill in grave cases, to use plentiful inunction with warm oil. When recovering from fever or any other severe illness, the native of Bengal always asks us, ‘Can I bathe?’ This question is not to be taken merely in its literal sense; it also means, ‘Have I recovered from my disease?’

TREATMENT OF TERAÍ FEVER AND OF OTHER PERNICIOUS FORMS OF MALARIAL REMITTENT.

Of late, quinine has been regarded as the chief remedy in these pernicious fevers, travellers generally taking a supply with them; as Macdonald, writing in 1843, tells us: ‘Those of some experience in these fevers never omit carrying a supply of calomel, which they use in large doses, often with the effect of checking the disorder.’ A military surgeon of great experience, who mentioned that he had treated about two thousand cases of bad remittent fever, writing in 1839,¹ stated that he had lost only about twelve patients. He appears to have been attached to the force of Sir David Ochterlony in their progress

¹ ‘In the Treatment of Jungle or Remittent Fever, of Hindoostan, Ptyalism is a Sign of Cure which is Rendered Permanent by Change of Air,’ *I. M. J.*, vol. vi. p. 773.

through the Nepal Terai. He remarks, 'My opinion is not singular that mercury in the treatment of remittent fever approaches as nearly as possible to a specific' . . . 'when ptyalism is produced.' He says, 'I was informed by an eminent medical gentleman, who observed sixteen cases of the terai fever at the verge of the forest as it runs along the range of hills near Almora. These cases were in the second stage and of a deep yellow colour; the first stage came on in the forest, so that they had taken no medicine. He considered these patients on the very verge of death; but yet, convinced, from what he had previously observed of the disease, that a bold measure was now the only means of saving them, he at once gave each *sixty* grains of calomel, and followed it up till ptyalism took place. Strange to say, he did not lose a man.' . . . 'I was informed by a young medical friend at Cawnpore that he had tried scruple doses of calomel five times a day with the most eminent success in remittent fever, both with Europeans and natives.'

Dr. Sullivan gives quinine and calomel freely in the *algide* pernicious state, as I did in the *convulsive* form. In the *comatose* form, when there is spasm of the pharynx or œsophagus preventing swallowing, Dr. Sullivan administers quinine by the rectum and hypodermically, as well as by rubbing in an alcoholic solution of the alkaloid, so as to cause absorption.

Webb's plan of treating the *algide* form, principally by calomel and opium, bleeding, and turpentine enemata and draughts, preceded by a hot bath (a very dangerous item of treatment in the collapse stage of cholera) for twenty minutes, was found so effectual that he could confidently trust it in the hands of the native doctor.

I was struck by noticing, six years ago, that Professor Fordyce Barker, of New York,¹ considers that Warburg's tincture is a far more effectual and speedy remedy in malarious fever than the largest doses of quinia. When I first went to India, more than one leading authority highly valued 'Warburg' in terai fever. Some considered, as Dr. W. S. Stiven did, that this agent owed its febrifuge power to *Berberis Lycium*, the famed *λύκιον* of the ancients, represented by the rusot of Indian bazars and its alkaloid berberite. Sir William O'Shaughnessy Brooke found the rusot useful in intermittent fever. In Dr. Morehead's hands, it 'proved unequal to preventing the paroxysm of ordinary intermittents.' Of late years, I heard little or nothing about it or Warburg's Tincture (which was considered an unpleasant agent). Indeed, to use the euphuistic phrase of one of my native students, it does not 'come within the chancery of my imagination' to rely upon any drug but quinia in this class of fevers when life is endangered.²

¹ *M. T.*, vol. i. for 1880, p. 327.

² For information regarding *Berberis Lycium*, vide Sir James Simpson on some Ancient Greek Medicinal Vases for containing Lykion, and the Modern Use of the

After the above was first written, I noticed¹ that Dr. Warburg communicated the formula of his tincture to Inspector-General Maclean in 1875. He states that quinine, in combination with a number of aromatics, is the active ingredient. Dr. Maclean adds his own high authority to the evidence of those who have found this tincture 'a remedy of great power in all malarial fevers.'

I need scarcely add that, if Dr. Warburg had not held the composition of his tincture a secret from the profession throughout my Indian career, I should probably have now been able to state that I had fully tested its efficacy in the treatment of grave Indian fevers.

For an injunction of great caution in giving this tincture in adynamic cases, see Waring's 'Manual of Therapeutics,' 4th edit. page 553.

Causes and Differentiation of the Remittent and True Enteric Fevers of India.—A few years ago, I, and probably any other physician of Indian experience, would have written upon the disease which we all concurred in designating as 'malarial or paludal remittent fever' with as little hesitation, regarding its etiology and diagnosis, as we should have felt in dealing with common ague. Now, however, the whole question has been torn in pieces, and there appear to be nearly as many conflicting opinions as there are professors. In fact, the opinions of Indian physicians regarding the true types of the grave fevers of that country are, just at present, in a state of transitional unsettlement, almost precisely similar to that which, more than forty years ago, Sir Thomas Watson described in his Lectures:—

'The mildest form of fever to which malaria gives birth is the intermittent or ague, but in climates and places where it exists in greater abundance and intensity, the fever becomes remittent, or even assumes the continued form. *This has led to strange errors—fevers which spring from the malaria, and which are never contagious, are confounded with the severe continued fevers usually called typhus, which are unquestionably communicable from person to person.*'²

Nevertheless, in India at present, this doubt is gradually resolving

same Drug in India; O'Shaughnessy's *Bengal Dispensatory*, p. 203; Dr. Kenneth Mackinnon, *I. A.*, No. v. p. 161. A friend of Dr. Mackinnon's wrote from the Neilgherries, 'I find that the medicine [Warburg's] is a favourite one with the Madras medical officers, who use it in all desperate cases, and with a success that is very surprising. —, who is travelling to join one of the regiments of the Hyderabad Contingent, had to pass through a jungly country, and caught a bad fever. He was for two days comatose and in a hopeless state, when the Warburg's Drops were given as a last resource. The effect was powerful and almost immediate, and he has continued to get on very well.' Dr. Stiven, *I. A.*, No. vi. p. 416; Dr. Francis, *Ibid.* p. 452.

¹ Dr. Maclean's article, Intermittent Fever, 'Quain's Dictionary of Medicine,' p. 735.

² The italics are mine.—N. C.

itself into certainty. That mysterious word, 'FEVERS,' which has always held so prominent but so dark a position in the Indian medical returns, is beginning to be intelligible to Indian physicians, who are able to perceive that it comprises intermittent, true enteric, and relapsing fever. So far the question is becoming clear, but what many of us still term 'malarial remittent' is now the subject of doubt and controversy. 'Is there,' it is inquired, 'any such thing as a paludal poison?' 'No!' 'Is there such a thing as paludal intermittent?' 'No; assuredly not!' We are told that 'the only intermittents are intermittent typhus, enteric, and relapsing.' Is there such a thing as paludal remittent?' 'No!' India is ravaged by 'continued' fevers; there is no such disease as 'remittent' fever. Sift and properly investigate your cases of the malady erroneously so called and muddled together in the official returns under that heading, and all 'remittents' will resolve themselves into cases of 'typhus, enteric, and relapsing fevers.' Others inquire, 'Does the true enteric fever of Jenner exist in India?' and reply: 'No; the so-called enteric of India is climatic, i.e. paludal remittent with bowel-complication.'

I have studied, with care and great interest, nearly everything which has been published during the course of this controversy. I think that the discussion has been most useful, because it has now reached the point at which the youngest practitioner in India must feel that his ignorance scarcely falls short of criminality if, after having studied all that the best Indian authorities have lately written on this subject, he fails, in those cases which he has watched throughout their full course, and examined after death, to distinguish which was Indian relapsing spirillum fever, which the true enteric of Jenner, and which the malarial or paludal remittent. To myself, the result of this pleasant course of reading is that, while it has greatly added to my own old store of valuable facts, it has left my chief and earliest opinions absolutely unshaken. One of these opinions is that, although marsh or jungle poison is the great factor in the causation of remittent fever, and although, in a very decidedly less degree, this poison may be everywhere a factor in the causation of enteric and relapsing fever, and probably often modifies their type when they occur in India, remittent fever is, etiologically, and in all its main characteristics, as distinct from these other two diseases as plague and ague are from one another.

As, although it still remains *sub judice*, the question of the differentiation of enteric and remittent fever in India has been much discussed, a few additional remarks upon the history of enteric fever, and especially of Indian enteric, may not be out of place here.

After I first wrote on this subject, I enjoyed the advantage of discussing it with Drs. Morehead and Maclean. What I heard from these great authorities fully confirmed the opinion which I submitted

to the profession in the columns of the *Medical Times and Gazette* in September 1879, which was that—although true enteric fever, or a fever closely resembling it, probably made occasional appearances in India between the times of Dr. John Clarke (1770–85) and of Twining (1835), and although Morehead saw two cases, and Scriven and Ewart each saw a solitary case, Edward Goodeve saw and described an outbreak of the disease in Calcutta, and I saw two distinct outbreaks and a few isolated cases in the same city—true enteric fever was long a rare disease in India until it became somewhat prevalent, in comparatively recent times, chiefly among European troops. No one who knew Dr. Morehead, and has, in studying his admirable work on Indian diseases, admired the rare combination of veracity and knowledge which every page displays, will imagine that true enteric fever ever prevailed to a great extent in his hospitals during the term of his long Indian experience. Before he went to India, Dr. Morehead learnt, while attending Louis' *clinique*, that there was a form of continued fever characterised by Peyerian disease. In India, he made post-mortem examinations in all his fatal cases of fever, opening the whole tract of the intestines. He appears, to my mind, to have met with and recorded a case of true enteric six years before Jenner separated this fever from typhus,¹ although, as Richard Bright had called enteric fever 'typhus with bowel complication,' Morehead called it 'typhoid remittent fever with gastro-enteritic symptoms.' While some of Jenner's most learned contemporaries died without admitting his discovery, Morehead accepted it early, when he wrote, in 1856, in his first edition, after describing case No. 32: 'While retaining this case in its original position, I must admit that recent inquiry may suggest that it was true typhoid, not adynamic remittent.'

I think it unquestionable that Bright and Morehead hold a very high position among those who almost succeeded in anticipating Jenner's grand discovery, nearly as Blackall, of Exeter, approached the demonstration of morbus Brightii.

In a valuable paper read before the International Medical Congress in August 1881,² Dr. Maclean writes:—'In the General Hospital at Madras, so far back as the year 1838, the year of my first connection with India, in the old infantry barracks at Secunderabad, in the Deccan, of dysenteric notoriety, and on service in China, as far north as Nankin, I saw and treated cases of continued fever, extending over twenty days in duration, with the bowel complication above referred to, that, without hesitation, if I saw them now, I would diagnose as enteric fever.'

¹ *Transactions of the Bombay Medical and Physical Society*, first series, vol. vi. p. 193.

² 'On the Prevalence of Enteric Fever among Young Soldiers in India, &c., *Trans.* vol. ii. p. 528.

The symptoms were the same, the mortality exceeded that of fevers distinctly malarial, they were not amenable to quinine freely given, and death from hæmorrhage from the bowels was frequent, and the intestinal lesions were what we now recognise as characteristic of enteric fever. It was on my return to India, after two years' residence in Europe, that I first satisfied myself that such cases, in all their essential particulars, were none other than enteric fever, as that term is understood among you here. I then saw, treated, and dissected cases as distinctly enteric in their symptoms, progress, and in their post-mortem lesions, as any to be seen in the London Fever Hospital. It is useless to add that all the attempts since made to persuade the public and the profession that enteric fever is not a disease of India have no weight with me.¹

In the discussion which followed the reading of Dr. Maclean's paper, Dr. Mouat, A.M.D.,² mentioned a very interesting case. He did not say where it occurred, but the context appears to show that it was in India. It was that of a young officer of the 38th Regiment, in 1840, which commenced as an ordinary attack of fever, ending in peritonitis, with perforation of the bowel. As there was suspicion of poisoning, Dr. Mouat made a most careful post-mortem examination, and found the now well-known characteristic morbid signs of enteric fever.

It is most improbable that there was then any considerable prevalence of enteric fever in India. I notice that, reporting in 1838, the elder Dr. Mouat, an officer of great Indian experience, said: 'Here' [in India] 'we have no typhus, and in no instance do our fevers appear infectious, unless the Palee fever or plague be an exception.'³

In our Indian career, Morehead, Maclean, Edward Goodeve, and I started with a full knowledge of the symptoms and post-mortem appearances of true enteric fever. We recognised it as a perfectly distinct entity in disease, although we gave it other names. I am confident that, unless by rare oversight on my own part, neither of us four overlooked cases of true enteric, which were as rare in our experience as Emperor butterflies are in that of European entomologists. The main battle of our lives was with malarial remittent; and, when true enteric came before us, we knew it almost as well as Jenner would have done, save that to us, or to me at least, its appearance was often unexpected, the presumption, at first sight, of a case in India, being:

¹ I am glad to find that my arguments against the, to me, absolutely incomprehensible doctrine that European soldiers, now serving in India, cannot suffer from pythogenic fever because they are not exposed to poisoning by drain or cesspool filth, receive powerful corroboration and extension in the facts and opinions on this point fully given in Dr. Maclean's memoir.

² *Transactions of the Congress*, vol. ii. p. 541.

³ *Madras Quarterly Medical Journal*, 1840.

‘ This is malarial remittent,’ the second thought only being : ‘ Or is it enteric ? ’ Five years ago, I said at the Epidemiological Society that, when I had a case of true enteric fairly under treatment, I could no more mistake it for malarial remittent than an old fisherman could mistake a dog-fish for a cod.

Deputy-Surgeon-General J. Marston, a very able physician, who, when Secretary to the Head of H.M. British Medical Department in Bengal, had the amplest information from the whole of British India at his command, has told us¹ that ‘ medical officers have latterly failed, in India, to satisfactorily trace out *the intimate connection of the disease with filth-causes of specific infection*, with which according to European authorities, it is invariably connected.’ Greatly as I esteem the authority of those who maintain the pythogenic origin of enteric fever, and freely as I acknowledge the striking character of the arguments upon which this doctrine has long rested firmly, I think that observation of the disease in India justifies a full reconsideration of the whole question in both countries. With this view, I submit the following—*not as a dogma, but as a mere tentative suggestion.*

Is pythogenesis the *prime, predisposing, and true cause of enteric fever* ? When I was young, this disease was prevalent on the continent of Europe, and comparatively very rare in England, where typhus was very common. I have already cited Sir James Paget’s mention² of the fact that specimens of typhoid ulcers, preserved by Hunter, who died in 1793, exist in the Museum of our College of Surgeons. Richard Bright was a middle-aged physician of great experience when he first observed it, and had no name to give it, late in the year 1826. Now typhus is rare in London, except in typhus years, and enteric fever is very common. In all times, but less at this moment than in any hour during the past millennium, cesspool and drain filth have tainted the air and the drinking-water of every city and country village in Europe. In their general bearing, the above remarks apply equally to India. To explain these facts in the history of this disease in the two continents, the opinion that its prime originative cause is an atmospheric or telluric influence—say a pandemic wave—now invading countries and now leaving them, travelling now broadcast, now in narrow lines, demands attention. Thus, in 1882, the inhabitants of Paris and our army in Egypt suffered from enteric fever outbreaks of unparalleled severity. All must admit that bad water was drunk both in the city and in the camp ; but I cannot think that these two great visitations of pestilence were merely coincidental. I believe that both were determined by epidemic influence. Convincing as nearly everything which I have seen in the writings of Dr. Billings is to my mind,

¹ Cited by Fayrer, p. 221.

² In the first Bradshawe Lecture at the Royal College of Surgeons.

I cannot bring myself to think with him that 'the term *epidemic condition* is simply another name for total ignorance.'¹ All my experience combines in persuading me that disease-waves are constantly flooding the air which we breathe. In clinging to this antiquated idea, much enlarged as it has been, in modern times, by Lawson and others—still greeting as a most welcome little stranger any microzyme which its discoverer may bring before me, as something really tangible, and worthy of having its morbid powers rigidly investigated—I am willing to admit that I and all who hold the same view are agnostics and visionaries. But the blind and deaf man, whose paralysed hand is dipped in water by another, and then held up to dry, must not deny that water and air are entities because they are not recognisable by his senses; and I am content to adhere to this somewhat dark faith until I can hit upon a belief which admits of demonstration.

With regard to sewer-filth, may it not be merely the most valid and prevalent *exciting* cause—not the *prime, true, and predisposing* cause of enteric fever? During the cholera season in Calcutta, almost any great imprudence, especially in diet, may excite an attack: not because that imprudence inoculates the system with cholera poison, but because, a general atmospheric or telluric influence having already placed the unknown prime cause of cholera there, the imprudence in diet, &c., is among those exciting causes which suffice to explode it, just as fulminate of silver is exploded if it chances to sustain a blow or shock, but may remain open on a plate for a week unaffected by other ordinary causes. Now, everyone in India recognises the fact that a dose of Epsom salts is, to the system doubtless already charged with cholera poison, nearly that which the blow of a hammer is to fulminate. A regiment of soldiers, eight hundred strong, in barracks at Fort William, pass through a cholera season with, say, half a dozen seizures, chiefly traceable to imprudence or exposure; but if, at any part of that endemic season, an enemy could give each of those men half an ounce of Epsom salts, I believe that at least seven hundred and fifty of their number would be down with Asiatic cholera within six hours. But no one would place Epsom salts among the *causes* of cholera. Thus, may it not be that—when the enteric fever wave covers us, and enervates and predisposes our system—water or milk containing sewage faecal matter is the most common and most potential *exciting* cause or explosive—all those who remain outside the verge of that wave, as well as many who are within it, remaining proof against this special mode of poisoning by drain and cesspool filth, as I believe we were long ago in England and in India, and may be again whenever this epidemic alters its course? Some may say, 'Further discussion is needless; this question has long been settled in England!' To this I reply, 'It will probably be reconsidered in India; and, what-

¹ *Transactions of Medical Congress of 1881*, vol. ii. p. 537.

ever the result may be, the inquiry will assuredly tend to advance our knowledge.'

I shall certainly not coincide in the opinions which may be arrived at by my brethren of the British Army if, at starting, they deny that, throughout the length and breadth of India, the European soldier, when on liberty in the bazaar, is liable to the calamity of quenching his thirst with draughts tainted by fæcal matter—nay, possibly by the excreta of native patients suffering from true enteric fever.

When, with abundant statistics and notes of post-mortem examinations at hand, medical men cannot agree whether an Indian fever is paludal remittent or true enteric, what I may term the PERFORATION TEST ought to come in as a useful aid. Twining, Ranald Martin, Morehead, John Macpherson, have all described lesions at the lower part of the small intestine in cases of what they regarded as malarial or paludal remittent. How came it, then, that they, Kenneth MacKinnon, Edward Goodeve, hundreds of other able Indian observers, and I, among the thousands of cases of malarial remittent which we treated and were prepared to write upon whenever we met with anything unusual, never saw or described a case of intestinal perforation occurring in remittent fever, this being an event looked for and dreaded in every case by all who know and treat true enteric fever?

A difficulty has been raised with regard to 'paludal remittent with bowel irritation.' When the first draft of this chapter was appearing, a very high authority asked me in writing, 'Does Peyerian ulceration only occur in specific enteric fever?' I replied, 'There may be follicular ulceration of (1) tubercular and (2) dysenteric origin; but, wherever I have found such' [i.e. Peyerian] 'ulceration in a case of *fever*, the other features of that fever have been those of true enteric fever.' A man with paludal remittent has no exemption from its congener dysentery, in which there is sometimes ulceration at the end of the small intestine; but no one who has seen the Peyerian ulcers of true enteric could possibly fail to distinguish them from dysenteric ulcers. If I saw the former in any case, whatever the life-history of that case might have been, I would at once declare, undoubtingly, 'This was a case of true enteric fever.'

After most of the above remarks were published, in 1882, Dr. Charles Morehead, a man whose admirable published works and noble personal character were universally esteemed by the profession in India, passed from among us in advanced old age, but without any appearance of bodily infirmity, and with a mind clear, wide, and deep—a fountain of living water, fresh and bright to the very last. Every word written and spoken by Dr. Morehead upon Indian disease is so valuable that I consider myself justified in citing the following passages from his then recent correspondence:—

Dr. Morehead's latest commentary upon the true enteric and other fevers of India.¹—The following was Dr. Morehead's reply to my inquiry as to his *present* views regarding the occurrence of true enteric fever in India :—

August 9, 1879.

'Our friend Grant has forwarded your note to me, and it has reached me this morning. I am absent in the country, away from all books, and shall be so till the 19th, when I return to Edinburgh; so that what I write to you, without delay, is from recollection, but yet sufficiently accurate, I believe, for your purpose.

'Refer to "Typhoid Fever" in the *second* edition of my "Clinical Researches," and to my report of sanitary progress in Bombay from 1830–1860, in the second volume of the 'India Office Sanitary Reports' (1868), head "Typhoid Fever," and you will find the degree to which the opinion in the first edition of the "Researches" is altered; and please do not, kindly, refer to me in connection with this question without carefully considering these references.² Now, in respect to what has been written of enteric fever in India since these dates—to the extent that I am informed of it—I would say, that the anticipations formed by me, as expressed in the concluding suggestions of the remarks on typhoid fever, in my *second* edition, of a probable epoch of confusion, have been realised; and I hope that the time is near when the subject will be cleared of its doubts and difficulties. I am not acquainted with any clinical report of enteric fever in India—I mean which gives the history of an epidemic, including *recovered* cases as well as fatal, *and* the ratio of deaths to recoveries; and until this is done the literature of the subject is manifestly incomplete. What I know of it in these later years is from post-mortem reports, chiefly those incorporated by Bryden in his statistical papers. I stated, in the suggestions above referred to, that we were not justified in concluding that where there was Peyerian ulceration there had of necessity been enteric fever. In addition to the exceptions mentioned by me' [at page 161, second edition.—N.C.], 'you will find in my chapter on Measles in the *first* edition of my "Researches," one or two fatal cases in which there was Peyerian disease, and, if I mistake not, Dr. Harley has somewhere also referred to this connection. Now, this pathological fact has been in great measure overlooked in these later years in India, and it has been almost universally assumed that the presence of Peyerian ulceration after death is evidence of there having been enteric fever during life. I believe it would not be difficult to pick out

¹ With this should be read an extract from a letter from the same authority to Sir Joseph Fayrer, at p. 170, *Climate and Fevers of India*.

² Reference to my chapter on True Enteric Fever in India (*M. T.* for September 20 and 27, 1879, pp. 336 and 363, and p. 121 of this volume) will show that this injunction was strictly obeyed.—N. C.

many of Bryden's reported cases, of which this is a forced and improbable interpretation. In many of them, after six or eight days' illness, there is extensive ulceration. This does not accord—I mean such morbid results after a few days' illness—with the history of enteric fever in European countries, and it ought not to be accepted of the disease in India, unless supported by undoubted symptoms during life, and the clinical histories of *coincident* recovered cases. On this point, however, I would remark that it is not improbable that it will be found, if it has not already been proved, that we may fairly look for modifications in the course of the disease as occurring in India, compared with this country, as, for example, in a more marked remittance, and in a more rapid course and a greater mortality, but still not to the degree which belief in Bryden's cases would imply.

'I cannot but think that writers on this disease, and on others common to Europe and India, are too dogmatic in formulating the etiology, the symptoms, and pathology too exclusively from observation in Europe, and that the history of many diseases, as observed in these two fields conjointly, has yet to be written. I feel sure that much of the obscurity, the contradiction, and difference of opinion rest on this, I would say, self-evident principle. I have one observation further—I cannot accept the statement so often put forth, that enteric fever was habitually overlooked by those of my generation of observers. I can say for myself, that I went to India familiar with the fact that, as described by Louis, there was a form of continued fever with Peyerian disease, and I, of course, followed the discovery made by Jenner some fifteen years afterwards, that this was a fever distinct from typhus; and it is not likely that, not working with my eyes closed, I should frequently have overlooked it, if it had come under my observation. There is a case of mine, in a girl of the Byculla Schools'—[this is Case 35, of Caroline Smith, at page 95 of the second edition. The observation (which Dr. Morehead cites here from memory, not having then his book to refer to) is, 'the glands of Peyer were distinct, and there were three or four round ulcers, each the size of a split pea; cicatrisation had commenced.'—N.C.]—'which has been several times referred to as an instance of this oversight; it will be found that the ulcers in the small intestines were cicatrised—evidence that they were the result of some previous illness, and not of the fever from which she succumbed. But it is useless to refer to particular cases, and I do not think that we are unreasonable in demurring to the verdict that we were ignorant and careless observers, and in continuing to insist that in our several fields of observation in India from 1830–60, enteric fever was rare, and that its clinical history in India since its greatest frequency has yet to be written; for assuredly during this period there has been, and there still continues, as I believe, much confusion and error on the subject of fevers in India.'

In a letter, dated two days subsequently, Dr. Morehead observes : ‘ One further remark before leaving this subject. In a prefatory note to No. 5, “ Medical and Physical Society’s Transactions, Bombay,” somewhere about 1843, I explained that it was my constant practice to examine all the organs, not only those expected to be diseased, and specially to open the entire tract of the intestinal canal, and I explain my several reasons for so acting. I mention this now as an additional reason for the confidence with which I maintain that I could not—except in a very occasional instance—have overlooked the existence of Peyerian ulceration in Indian fevers.’

The following observations occur in a commentary upon my chapter on the true enteric fever of India, which Dr. Morehead most kindly sent me. I had remarked : ‘ When Dr. Morehead published the first edition of his treatise in 1856, he believed that this malady’ [enteric fever] ‘ did not occur in India, although the type of disease in some of the cases which he recorded closely resembled that of enteric fever—a fact which he clearly observed, when, after describing Case 32, he added : “ While retaining this case in its original position, I must admit that recent inquiry may suggest that it was true typhoid, not adynamic remittent.” ’ Upon this Dr. Morehead remarks : ‘ At page 337¹ you rightly interpret me, with this slight exception : I do not know that you quite recognise that it hangs exclusively on one Case (32). Therefore, instead of “ the type of *some* of the cases,” it should read “ *one* case.” It was that of a man of the 15th Hussars, quartered for some months in the town barracks of Bombay, the sick being sent to the General Hospital under my care. It was, therefore, if *enteric*, a sporadic case in a body of men exposed to the same influences ; therefore, taken in its connection, it proves little as to the existence of enteric in Bombay in those days (1840). Case 35 is another which I am said’ [not by me—N.C.] ‘ to have mistaken. A girl, after eight days of febrile symptoms, dies ; at the end of the ileum were three or four small ulcers the size of a split pea ; *cicatrization* had commenced. Could this lesion be regarded as the result of the eight days’ febrile condition, or could it be regarded otherwise than [as] an antecedent lesion ? And yet, on these two cases, in the first of which, when first reported in the “ Transactions of the Medical and Physical Society,” I remarked on the appearances as resembling those of the European continued in one of its forms, and this six years before Jenner wrote—it is affirmed that I was in the habit of overlooking enteric fever and calling it remittent.

‘ Then further note that the case which I first observed in 1856, and which showed me that enteric fever was to be admitted, occurred to me before I had seen the reports of Ewart, Scriven, Goodeve (“ Clinical Researches,” second edition, page 160). Could you have

¹ [Vide the corrected passage at p. 127 of this volume.—N.C.]

stronger evidence that I had not been purblind during the thirty years of my Indian practice, and that it would be quite as reasonable for European physicians to maintain that my not having witnessed scarlet fever in India was simply an error in diagnosis from having mixed it up with measles, with which I was familiar, as physicians in Europe had done—before Home, some hundred years ago? Medicine can never progress as a science if we cut the Gordian knot of difficulties by assuming ignorance and incompetency in the observers who have preceded us, unless the indications are so manifest as not to be evaded. I entirely agree in the very excellent remarks which you make as to the course which ought to be pursued in the future. I do not pretend to have seen the greater number of the reports which have come from India, as ' . . . 'for these are not accessible to outsiders, but I have seen sufficient to convince me that there is much looseness and inaccuracy and want of the close method and precision which clinical observation and record require before they can be received with perfect confidence. In a word, the hobby has become rampant and run away with its riders, which, as you observe, is exactly what I predicted, but it has occurred to an extent I could not have ventured to anticipate.

'One word of doubt. I am not sure that you interpret rightly the reference which you make at page 386 of your paper [pp. 123-4 of this vol.], antecedent to "Twining"' [I give a citation from Annesley, which Dr. Lyons holds to be evidence that enteric fever was observed by that authority in India.—N.C.]. 'To me the incident seems to resemble what I describe at page 24, "Clinical Researches," second edition, beginning at "the mortality," and on to the top of page 28' [intermittent complicated with diarrhoea or dysentery.—N.C.], 'much more than anything allied to enteric, and I am quite sure that these were not enteric.'

Seeing that the whole question of the etiology of Indian enteric fever is at present quite unsettled and subject to investigation, I propose the following considerations, results of my experience—not as dogmatic conclusions, but as texts for inquiry.

Only one form of true enteric fever has occurred within my observation in India.

The morbid appearances of Peyerian disease in true enteric are so characteristic that no morbid anatomist can fail to distinguish them from all other ulcerative and inflammatory lesions to which the ileum is subject.

If I found these characteristic appearances in an ileum, I would declare the case to be one of true enteric, quite irrespective of its life-history, just as I would distinguish variola by looking at its fully developed eruption.

The follicles of the ileum are liable to dysenteric and tubercular

lesions; but, whenever I have met with ulceration here in a case of fever (hectic excluded), the other features of the disease have been those of true enteric fever.

I have repeatedly seen true enteric fever in Calcutta having all the characteristics of the enteric of Jenner. I have seen it only in Europeans and Indo-Britons. I believe that its prevalence among natives has been exaggerated; but, of course, I am not justified in giving a dogmatic opinion upon that which I have never seen. I believe that it is frequent among young European soldiers, but I think that this matter still requires the test of strict future observation.

I recognise the enteric fever of Jenner as an entity so distinct that I would not deny the identity of a case in which I found all its clinical conditions, only because I could not discover that it was due to this cause or to that, (pythogenesis or what not,) any more than I would ignore the existence of a man standing before me only because I failed to discover the name of his grandfather.

In India, I would approach the investigation of a so-called case of true enteric fever with the very maximum of scepticism compatible with belief in the rare occurrence of the thing looked for and an honest desire to elicit truth.

Unless the case ended in perforation, I would not accept, without question, the assertion of any professional brother that he had under his care a case of Indian enteric fever, unless he convinced me that, in symptoms and progress, and, if fatal, in morbid appearances, it had all the leading characteristics of English enteric.

Malarial remittent, more or less amenable to quinine, uncomplicated with looseness of bowels or with follicular disease, is one of the most prevalent and fatal diseases of India. Malarial remittent may be complicated with, or followed by, diarrhoea or dysentery. But there is nothing to show that such complicated cases of marsh or jungle fever are characterised by the Peyerian disease of true enteric fever.

As regards their probable modes of origin, I draw the strongest line of distinction between malarial remittent and enteric fever. The former is due to malaria solely. May it not be that, like plague, enteric fever is epidemic, and that attacks are *excited*, not *caused*, by the ingestion of sewer-filth? The facts that it is not subject to the antidotal action of quinine, and that its existence cannot be discovered in many localities where the marsh poison is most virulent, make it probable that it is not generated in marshes. But there are grounds for considering that it can only exist in an atmosphere which, like that of London or of Calcutta, is pervaded by marsh exhalation.

Marsh and jungle malaria of high intensity may somewhat modify its type.

It is now too late to question whether decomposing sewage matter, when taken into the system, acts as a specific poison producing the fever, or as a mere excitant and explosive?

It is certain that the term 'typhoid' has led to so much confusion, in the writings of careless people who ought to have known better, that it ought to be stamped out of medical nomenclature, the designation 'enteric fever of Jenner' taking its place.

EPHEMERAL FEVER—COMMON CONTINUED FEVER— ARDENT FEVER.

There are very few who do not suffer, within a few days after landing in India, from some form of what used, in old times, to be called 'seasoning,' or 'acclimatising' disorder. The minority are attacked with dysentery or Asiatic cholera; the majority pass through a less severe ordeal of catarrh, or diarrhœa, or fever. The fevers to which new arrivals are especially liable are febricula or ephemeral fever, the febris continua simplex of the College of Physicians' nomenclature, which formerly appeared in the Indian army medical returns as 'Common Continued Fever' or 'Febris C.C.,' and Ardent Fever.

Physicians have never been agreed with regard to the true nature of these fevers, and I believe this question to be as much open to controversy to-day as it has ever been. Thus, although these fevers may appear in temperate weather, they are especially prevalent in hot climates; but is the moderate ardent fever which now shows itself during the hot dry weather preceding the rains in Lower Bengal the deadly malady described under that title by Twining in 1835? ¹ Again, are the febricula, continued fever, and ardent fever of, let us say Bermuda, Aden, and the hotter and drier parts of India, identical with these diseases in Bengal? Still again, was not much of that febris C.C., which used to figure so largely in the medical returns of European troops serving in India, malarial remittent, neglected intermittent, true enteric or relapsing fever, or some or any imperfectly differentiated fever which the observers of fifty years ago may be pardoned for having ranged under this indefinite but very comprehensive heading, seeing that the London College of Physicians were formerly content to define simple continued fever as 'continued fever having no specific character' and, in the latest edition of their nomenclature, speak of it as 'having no obvious distinguishing character'? Certainly, within my own time, the ideas of many otherwise well-informed Indian practitioners upon this subject were at once loose and discordant. Many years ago, the charge of certain wards was equally divided between an able and experienced brother officer (now deceased) and myself. Having occasion to look over a vast number of my colleague's bed-head tickets, I exclaimed to myself in surprise: How comes it that he has treated hundreds of cases of febris C.C., and that my tickets do not

¹ *Op. cit.*, second edition, vol. ii. p. 235.

contain one of these entries? Indeed, during the whole of my Indian service, I never recorded a single case as common continued fever or ardent fever. A few years ago, I asked an admirable physician who served with European troops throughout the Burmese war of 1852-53, during which deadly fevers were extremely prevalent, 'What used they to call febris C.C.?' He replied, with a smile, 'The men used to run about in the sun and get fever, which they used to call common continued fever.'

Writing in 1865,¹ the late Dr. Chuckerbutty observed: 'Sixteen years ago, there was a great tendency to inflammation of important organs in the course of remittent fever, and hence the heavier rate of mortality then than now from this disease. The common continued fever, too, counted its victims in those days, whereas one seldom hears of anybody dying of it now. This accounts for there being no deaths from it in the second decennial period commencing 1860; while it had a mortality of ten per cent. in the first, commencing 1850.'

I believe that I have always, since the occurrence of my first case, been well acquainted with the cause and nature of the febricula, simple continued fever, and ardent fever of Bengal (I know nothing, by experience, of these diseases as they appear in other localities); but, strange as at first sight the assertion may appear, I have no doubt that the ease with which I learnt this important lesson was due to the fact that I was absolutely unacquainted with the abundant but most confusing literature of these maladies until many years after I had been in the constant habit of encountering them in practice. I probably owe my success and freedom from great error in this direction to my late dear and accomplished friend and fellow-student Joseph Richard Bedford. When I relieved Mr. Bedford of the medical charge of Chittagong, a few months after my arrival in India, he gave me, in a few words, an admirable sketch of the prevalent diseases of that locality. He said that the most prevalent fevers were of paludal origin, and that they were subject to cinchonism. My own observation speedily confirmed his; and, as I did not see true enteric fever until I had served for many years in India, I treated all fevers, with the exception of the exanthemata, with quinine—the plain result of which is that, from that day up to the time of my retirement, *no case of simple continued fever or of ardent fever ever fully developed itself under my observation*. I repeat that I can say nothing from experience of the common continued and ardent fevers of other parts of the world—as, for example, the disease described² in 1879 by Surgeon-Major Green as occurring at Aden during a recent cold season, in which, between January 7 and March 1, the evening temperature of a patient was never below 100° except on one (late) day, and, during the first four days,

¹ 'A Clinical Retrospect, &c., I. A., No. 21, p. 109.

² I. M. G., July 1879.

ranged from 104° to 105°. Probably the type of the disease varies with the climate and with the presence or absence of the paludal poison ; but assuredly he who, in Bengal proper, treats all cases of ordinary fever (excluding jungle fever, enteric fever, and the exanthemata) with quinine as soon as perspiration shows itself after the first paroxysm, will find that febricula is a very ephemeral disease indeed, and that his experience of common continued fever and ardent fever is extremely small. I am confident that these three diseases, or, to speak more accurately with Dr. Morehead, these three degrees of the same malady, are, in Lower Bengal, the most usual types in which intermittent fever first attacks the robust, full-blooded, and perhaps free-living European who arrives there during the hot weather.

I know no more useful exercise to the mind of a young Indian physician than will be afforded by a careful perusal of the fifty pages in which William Twining records his experience of the continued or ardent fever of Bengal. Up to the time of his death, no Indian physician ever viewed disease more carefully, more honestly, or with more science, according to the light of his day, than Twining did ; but, in all probability, nothing that stands recorded upon the shelves of the Medico-Chirurgical Society's library has ever led to so much etiological error, false practice, and sweeping mortality, as the facts and arguments embodied in this well-digested chapter by this truly eminent authority have done. Making allowance for the probability that marsh miasm was more intense in Calcutta in Twining's time than it has been of late years, and that, consequently, the intermittent of the hot season was more severe, it appears to me that all the error in his theory and practice arose from the fact that he did not try his cases of ardent fever by the QUININE TEST. Had he done so, instead of vainly endeavouring to subdue the malady by copious bleedings and tartar emetic, his 'ardent fever' would never have become 'continued fever'—it would have been cured after the first paroxysm, and would have appeared in his hospital returns, as it invariably did in mine, as intermittent fever. What the intermittent fever of the hot weather in Bengal would become if we did not stay it promptly by quinine, I do not know ; I often wished to know, but I did not venture to expose any patient to the danger of the experiment ; but it is probable that a case, so neglected, would, in a few days, become a fair example of Twining's 'continued' or 'ardent' fever. It will be seen that quinine entered into the treatment of some of Twining's cases of ardent fever, but its use was subordinated to that of copious V.S. and tartar emetic, and was only had recourse to in doses of from two to four grains when, as he expresses it, 'a remission of pyrexia afforded room for the employment of quinine.' Those who combated Indian fever by blood-letting committed disastrous havoc by the fatal error of treating that mere name 'ardent fever,' instead of that true entity the paludal inter-

mittent of the hot weather. I never had recourse to V.S. in any case of Indian fever, however severe the head symptoms might be. In one case, soon after my arrival, I applied leeches to the temples. I felt convinced that they did no good, and I never considered it needful to employ this treatment again. Twining nowhere ascribes ardent fever to the paludal poison; neither does Morehead, who speaks of this disease as being most prevalent in parts of India where 'the conditions of malaria are absent.'¹ To Kenneth Mackinnon is due the credit of having ranged ardent fever with paludal remittent, attributing both to the same cause—the marsh poison. He writes²:—'Authors on Indian diseases have usually divided fevers, according to the season of the year at which they occur, thus: the ardent fever of the hot weather, the bilious fever of the rains, and the congestive fever of the cold season. The first and last it is usual to call continued, and the second remittent, although all three may assuredly be produced by the same remote cause. This may be illustrated by the observed effect of exposure in localities (the forests of Singhboom, for instance) where at all periods the miasmatic cause of fever is rife. That heat alone, and specially solar exposure, may cause fever, independently of malaria, seems a fact admitted on all sides; but we shall often have difficulty in deciding (when fever is prevalent in the hot months) upon which of these causes the prevalence depends. I have elsewhere alluded to our defective knowledge of the whole conditions essential to the generation of malaria, and I have noticed the fact that it may undoubtedly emit from dry surfaces; nor are we to forget that showers frequently fall in the hot months, immediately followed by a hot and dry atmosphere, endowed with wonderful evaporating power. It must also be remembered that in some parts of the country, even during the hot months, there are extensive surfaces which only then are becoming dried up. In these the ardent fever of the hot months will have some character in common with marsh fever; and in seasons when rain falls, according to my observation, the hot-month fevers will be found to exhibit a periodic tendency even in the open localities where our cantonments and civil stations stand.' Mackinnon devotes sixteen closely-printed pages to the treatment of ardent fever, under the heads of Bleeding, Use of Cold Water, Purgatives, Mercury, Sedatives, Quinine, and Diet. A perusal of these pages affords an admirable example of an original but most cautious mind struggling to free itself from the trammels of tradition and authority. Thus he commences by saying that 'it is in this type of fever that we may use the lancet with the greatest freedom and safety.' But, immediately upon this, the wise and observant spirit of reform enters and prompts him to write, with only the break of a semicolon, 'but I must yet express my conviction that, even in this disease, it is used too indiscriminately in Indian practice, because I

¹ *Op. cit.* p. 162.

² The Treatise, p. 210.

have observed that blood-letting often produces a lasting injurious impression on Indian residents. New blood is not easily made, and it has appeared to me that there is an irritability of constitution and a tendency to local determination caused by the want of blood in proper quantity and quality. I am only arguing that there are many cases of ardent fever where general bleeding may be dispensed with.' Still, his general treatment of this disease, by bleeding, tartar emetic, strong purgatives, mercury, and opium, is decidedly heroic, and I would not dare to follow the directions for the use of quinine which close his dissertation upon his 'sheet anchors' named above. 'I make it a rule,' he says, 'to give quinine in all fevers after the febrile symptoms are gone. As already hinted at, we may, in this fever, notwithstanding its usually continued character, have at times decided remissions and exacerbations. When the remission is well marked, and the complications are not serious, quinine may be given; but we may here wait more for almost if not for complete apyrexia; and I believe it is also correct to say that it is more apt to bring on heat and local determination, if given in this fever too soon, than in regular marsh fever, but there is not so much necessity for using it, and it may be given in smaller doses.' Here this eminent authority is not free from the weakness which was general when he wrote, and which has, unhappily, again prevailed largely of late years especially among native practitioners in Bengal, of not daring to give quinine, in periodic fevers, until all heat of skin and headache are arrested. Just as Mackinnon wrote this, Indian medical men began to perceive, mainly in consequence of the facts and arguments of Drs. Hare and A. C. Macrae,¹ that we can administer quinine without risk and with excellent effect in severe cases of paludal fever while headache remains and the skin remains hot. In cases of the ardent fever of the hot weather, and in all other serious cases of paludal fever, I gave quinine as soon as a little moisture could be felt behind the ears or in the inside of the wrist. I never found it interrupt the advance of defervescence. In an ordinary case I gave one six-grain dose of the alkaloid with a quarter of a grain of ipecacuan, to be followed by three-grain doses of quinine every three hours, and a double dose at any sign of the return of fever. In cases of ephemeral fever or febricula, I kept the head cool and used ipecacuanha freely during the hot stage, and gave quinine in the above-mentioned doses immediately upon the appearance of perspiration, never neglecting that free but moderate purgation which is indispensable in all cases of malarial fever. I very rarely saw pyrexia recur in any case so treated. I may here give two cases which are fairly illustrative of the fevers now under consideration.

The first was one of severe febricula, such as is frequently seen among sailors in the port of Calcutta during the heat-stroke weather

¹ *Indian Register of Medical Science*, 1848, p. 360.

which precedes the rains. A handsome, fresh-coloured, young seaman was brought in with strong fever, flushed face, congested eyes, and very severe headache. I said to him, 'Your skipper has made you work over the ship's side in the heat of the day?' He replied, 'Yes.' Knowing what often occurs in a day or two after a ship arrives after a long voyage, and seeing that the insides of his hands were tarry and the outsides red and blistered, I had no difficulty in recognising the cause of his attack. Captains of the lower class, being unwilling to employ native painters, compel their men to smarten up the sides of their shabby, weather-stained crafts. A more dangerous position than that of a full-blooded sailor hanging for hours, in heat-stroke weather, between the torrid sky and the glassy river can scarcely be imagined. We had some little trouble with this man's case; but, by keeping his head cool, giving him plenty of ipecacuan and salines, throwing in quinine immediately perspiration followed vomiting, and purging him freely, he steadily recovered.

Sixteen years ago, I was sitting at office work in my quarters at about mid-day, experiencing certain moderate but very definite head-symptoms which always warn me that the day is one upon which we may expect cases of heat-stroke, when I was called to visit our house-surgeon, who had been taken suddenly ill. Dr. — may be described as an excellent specimen of a well-conditioned young Scotch gentleman of rather sanguine temperament, not long resident in India. He was partially insensible, but complained of his head, and had the dry and burning skin of insolation. Although I had seen a great deal of heat-stroke, I never doubted that this was a genuine case; and, as I acted, I reflected, as I once did in the case of a young native whom I treated for heat-stroke: 'Under what law of nature am I, a by no means strong man, allowed to minister to this healthy-looking youth, thirty years my junior? Why does the heat which has struck him down spare me?'

My treatment of Dr. — chiefly consisted in rubbing, as if planing, his unshaven scalp with smooth masses of ice. This was continued for hours, until after sunset, when he began to express relief and to perspire. I then at once saw that the life of Dr. — had been placed in imminent peril by an attack of intermittent fever, which happened to make its first onset in heat-stroke weather. He took quinine, ipecacuanha, and a purgative, and recovered speedily without another bad symptom. Had his case been neglected, he would, I believe, have died that afternoon, and the death would have been registered as one of 'sun-stroke.' Had he been freely bled and leeches, and drenched with antimony, mercury, and drastic purgatives, he would have probably made a slow recovery from a typical attack of the 'ardent continued fever' of the old writers.

MALARIAL CACHEXIA.

It is certain that many of the gravest diseases of India, not generally classified with those which are directly produced by malaria, are largely due to this cause. Among these may be numbered Asiatic cholera, dysentery, suppurative hepatitis, land scurvy, sloughing of the cornea, menorrhagia, a tendency to abort, post-partum hæmorrhage, puerperal fever, tetanus, chronic muscular and articular rheumatism, pneumonia, and certain prevalent forms of diarrhœa. Morehead says: (p. 25)—‘There can be no question that much of the mortality attributable in India to “bowel complaints” is, though indirectly, yet chargeable to the account of malarious fever.’ This just remark has, I am satisfied, application to that very prevalent form of deadly chronic intestinal flux which is so destructive to the lives of ill-fed Bengalees that I have termed it *Morbus Bengalensis*. Hence much of the inclination of some of our first authorities to conclude that, in India, true enteric and relapsing fever are wholly due (as they probably are in part) to ‘climatic causes’—i.e. to malaria. It would scarcely be an exaggeration to affirm that this malarial cachexia is, more or less, the acquired constitutional state of everyone who has resided long in India. Everywhere in that country, and in the persons of all old Indians there and at home, the malarious taint is detectable in almost every kind of disease. In this chapter, however, I shall confine my remarks as much as possible to those conditions of disease which either follow malarious fevers in India or are more or less directly traceable to the operation of malaria in that country.

Splenic Hypertrophy, so well known to those practising in the malarious districts of England as ‘ague cake,’ is of great but exceedingly unequal prevalence throughout the larger portion of the plains of India. The occurrence of splenic hypertrophy, as a result of paludal fever, is ruled by laws of which we have no knowledge. It appears to depend not upon the type or severity of fever, but upon peculiarities of locality and race. Thus, as we shall presently see that elephantiasis prevails only in marshy districts within the range of the sea breeze, and that, where it is most prevalent among natives, it very rarely attacks Europeans—so splenic hypertrophy, while it is not generally prevalent among Europeans, is most common among natives in the plain country of Bengal and Bombay, but is comparatively rare in the Madras Presidency. Thus, in *Bombay*, Morehead found that, out of 243 clinical cases of intermittent fever, enlargement of the spleen was present in 91. The infrequency of this lesion in the *Madras* Presidency was noticed by Annesley. In the Deccan, during five years ending June 1855, in an average strength of 10,500 men, there were 15,684 admissions for fever, which were generally amen-

able to quinine. Out of these, there were only thirty-nine for splenic disease, with two deaths. Dr. Francis Day adds that in none 'of his own cases of fever in the 3rd Regiment of Infantry, Hyderabad Contingent, at Bolarum, could enlargement of the spleen be detected.' Doubtless the general immunity from this lesion enjoyed by the European inhabitants of malarious districts is, in a large measure, due to the promptitude with which their attacks of fever are encountered and to the superiority of their hygienic surroundings. Still, this is far from affording a full explanation of the difficulty, seeing how great has been the prevalence of 'spleen' among Europeans in certain localities. It has been the leading characteristic of Peshawar fever among our European troops. This condition was almost inseparable from Chittagong fever in natives; but, during a residence of more than three years, I never saw it there in a European. Although, as I have said, I believe that I had splenic enlargement when a child in the Kentish marshes, I have never, in thirty-six years' suffering from Chittagong and Calcutta fever and their sequelæ, experienced the very slightest evidence of splenic uneasiness or enlargement. Splenic hypertrophy is very common among natives in and around Calcutta, but I do not recollect ever to have seen a European who got 'spleen' there; whereas, at Mutlah or Port Canning, forty miles from Calcutta, some five days' fever brought the spleen down in robust European sailors just come in from sea. I should recognise a recent Mutlah spleen now. It appeared like a goose-egg below the ribs in front. One of my patients, a magnificently developed gigantic young northern sailor, died from rupture of such an acutely enlarged spleen, this lesion being, apparently, quite spontaneous.¹ When Peshawar fever and spleen were most prevalent, thirty years ago, among our European soldiers, the native inhabitants of the Valley were not at all remarkably liable to these diseases. Otherwise, my own personal experience of 'spleen,' in well-to-do Europeans, is very small indeed. I can only recall two cases. One of these occurred in Bengal, the other in the N.-W. Provinces. Both the sufferers were officers enthusiastically devoted to the pursuit of game in malarious localities.

I need scarcely add that, whether our patient be a native or a European, we neglect his case if we do not examine the spleen at every stage of malarious fever.

In 1845, Government received so many reports of the prevalence of fever along the line of the Delhi Canal, that they appointed two engineers and a medical officer to report upon the health of the inhabitants of that large portion of the N.-W. Provinces which was under

¹ This poor fellow suffered from a very noteworthy symptom—an unceasing call to empty the bladder. I also saw this in the case of Chief Justice Norman, who died from stabs in the belly, the cause being pressure by blood collected in the pelvis.

irrigation. It was suggested by the medical member, the late Dr. T. E. Dempster, that the condition of the spleen in any given number of individuals would be a fair test of the frequency and degree in which they had suffered from malarious influence. This test was applied as follows:—At each place, twenty children (it being believed that young persons are more liable to enlargement of the spleen than adults) and twenty adults were selected, the chief care being to take a fair sample, not of the sick, but of the ‘going about’ population. The Committee travelled about 1,400 miles, visited more than 300 inhabited localities, and examined upwards of 12,000 individuals of all ages in both irrigated and un-irrigated districts. It was found that, in places irrigated from the Western and Eastern Jumna canals, the percentage of enlarged spleens ranged from 16 to 59 among adults and children of all classes, the proportion of instances in which the percentage was 25 and upwards being as 8 to 5 of those in which it was below that rate. In lands irrigated from wells, in the high land of the Dooab, only 8 per cent. of enlarged spleens were found; in un-irrigated countries the percentage ranged from 3 to 44; in naturally malarious localities, from 34 to 44.¹

Thus originated what has ever since been known as *Dempster’s spleen test*, which can never be neglected without danger when new ground has to be taken up for Indian cantonments, &c.

The association of *splenic disease and scorbutus* has been often observed in India and elsewhere, and in both sea and land scurvy.² As I have already stated, the latter disease is of great prevalence in India generally, and especially in certain districts, as in Behar and Scinde. It would be very interesting if officers on tour through such districts would observe and report how far scorbutus and spleen are associated in their inhabitants.

So common among the poor in India is splenic disease, and so fragile is the organ, in many cases of men quite capable of performing their daily work, that it might almost be laid down as a rule that to beat or kick a native is to endanger his life (*vide* my ‘Medical Jurisprudence for India,’ pages 457–67).

Immense spleens are daily met with in most of the native hospitals

¹ ‘Notes on the Application of the Test of Organic Disease of the Spleen as an Easy and Certain Method of Detecting Malarious Localities in Hot Climates’ (1848); and an additional communication, *I. A.* for January 1858, p. 293. It was found that the prevailing sickness was not due to irrigation, but to the canal works or the watercourses of private individuals having intercepted the natural drainage of the country; and having thus led to the formation of swampy tracts, diffusing malarious influence around them.

² See a valuable paper by Surgeon-General W. A. Green, ‘Cases of Sea Scurvy, of Scorbutic or Putro-adynamic Fever, and of Fever complicated with severe Spleen Disease; with Remarks to point out severally a Similarity in their Probable Causes, in their Nature, and in some of their Symptoms,’ *C. M. T.*, 1839.

in the plains of Bengal and Bombay. The extreme limits of enlargement are from about the level of the sixth rib, when the lung and heart are pressed upwards¹ from the spine to the right of the umbilicus and down to Poupart's ligament or below it. In examination, we percuss the left chest, and then, the patient lying down, we place the open hand, in front, over the left hypochondrium, where the mass of the enlarged organ is felt. If there be ascites, we give the spleen sudden but gentle pressure backwards with the forefinger. It generally sinks in the fluid, touches the spinal column, and immediately rises again, striking the finger sharply, as in *ballotement*. We then run the finger across the tumour at its broadest part until we feel an abrupt rounded edge, when we easily trace its very defined inner margin, often from the scorbiculus cordis to the pubes. We must be careful to do all this with a perfectly light touch; these spleens are often too brittle to bear any 'punching' and rough handling.

The following is a description of the condition of an hypertrophied spleen in the museum of the Calcutta Medical College, given by Dr. Joseph Ewart in his admirable Catalogue:—

'Specimen 547. Enlargement of the Spleen from Malarious Fever.—The peritoneal covering and capsule are much thickened and puckered from organised inflammatory exudation. In the depressions caused by this contraction, the adventitious tissue exists in greatest abundance. The capsule and peritoneum, as they are seen on section inseparably united, measure about a line in thickness. The trabeculæ are thickened and very distinct, the interspaces large, and filled with the spleen-pulp. The density and compactness of the organ is greater as we examine it from centre to circumference. About an inch of the periphery is so compact that the trabecular spaces do not exceed the diameter of a pin's head, whilst many of those towards the central region of the spleen are large enough to contain a pea.'

Where the spleen slowly enlarges under repeated attacks of fever, inflammation probably has little or nothing to do with originating the process; such inflammation as occurs is intercurrent. But many enlarged spleens present such marked traces of inflammation externally, peritoneal opacity, contraction and thickening, adhesion to the diaphragm, and to other adjacent parts, and inflammatory sub-capsular deposit, that we cannot doubt that *splenitis* has been present. The spleen may be very tender under pressure when its enlargement has been sudden, and in many cases there has been local peritonitis. Probably this inflammation of the splenic peritoneum occurred in some at least of the splenic cases at Peshawar mentioned above (pp. 196 and 222), and gave a colouring of reason to the practice of leeching which I have described.

¹ Morehead, p. 36.

Splenic Abscess.—Suppuration, leading to the formation of distinct abscess of the spleen, apart from pyæmic infarction, is a rare condition in India. Preparations Nos. 554 and 557 in the Calcutta Medical College Museum appear to illustrate this lesion. Dr. A. A. Mantell narrates¹ a case in which a very large splenic abscess was evacuated through the left lung; the man, a European, aged sixty-two, had been long employed in India as a pilot and as the superintendent of a lighthouse on the Cuttack coast. A history of fever is not clearly made out; but, as there was an abscess in the throat, and another in the right kidney as large as a hen's egg, it is not improbable that the disease was tubercular. Dr. Verchere reported² the case of a man who had been ill for a considerable time at Loodiana with fever. He complained of great pain in the left side, and the spleen was acutely tender and 'enlarged to the size of a large fish.' It was leeches and blistered. A few days later, fluctuation could be felt in the spleen, and the pain was intense; poultices were applied, and, as soon as an indistinct pointing appeared, the abscess was tapped with a hydrocele trocar, the canula being left in for some days. He made a perfect recovery, and went to duty without being ill or inconvenienced. The general health improved much, and the man, who had been emaciated, feeble, and livid-grey-complexioned, soon made flesh and assumed a healthy appearance. At nearly the same time, the civil surgeon, Bhawulpore State, recorded³ the very important case of a young native man who had suffered from fever and enlarged spleen about ten months previously. The spleen was acutely tender; it extended beyond the mesial line of the abdomen and downwards nearly to the crest of the ileum. The entire left chest was evidently full of fluid. An opening was made in the left iliac region; only two ounces and a half of clear pus escaped, and the distressing symptoms were not relieved. There being pointing above the seventh rib, a trocar was introduced, and one pound thirteen ounces of healthy pus were drawn off, with marked relief to the distended chest. Next day, one pound four ounces more were removed through this aperture, which made a decided impression on the bulk of the spleen. The thoracic and abdominal cavities were, therefore, in communication. Inoffensive pus continued to flow, to the amount of six pounds fifteen ounces, during the following fourteen days, when the patient was taken away by his friends—a most common termination (as far as we are concerned) to our most interesting cases in India. The compressed lung rapidly expanded, and the heart's sounds were heard to the left of the sternum. When the patient was last seen, he was free from suffering, respiration was tranquil, air was heard all over the left lung, the spleen was much reduced, there was no hectic, and he was making flesh.

¹ *I. A.*, No. xviii. for 1865.

² *I. M. G.*, June 1869.

³ *Ibid.*, October 1, 1869.

Dr. Chandra has given¹ a case of abscess of the spleen which was successfully treated by free drainage.

Dr. W. H. Bull has reported in the 'Lancet'² the very interesting case of a groom, aged forty-two, who had served for nine years as a soldier in India, and had suffered there from hepatitis and from several attacks of fever. Twelve years after his return home, he died from repeated attacks of hæmatemesis, caused by perforation of the stomach involving a medium-sized branch of the splenic artery. This mischief was caused by an abscess, apparently splenic, not larger than a small orange. The lower part of the spleen was 'entirely disintegrated and in a state of ulceration, forming the outer boundary of the abscess wall.'

Specimen No. 556 in the Medical College Museum is one of *Gangrene of the Spleen* in a European who had been suffering from intermittent fever.

I never saw *gangrene of the spleen* in practice, but we read of a disease which, in 1831, was called the *Siberian epidemic*, or 'splenitis gangrenosa,' which appears to have been a pernicious fever, often ending in 'symptoms of putridity, severe pain in the side, meteorismus, excessive thirst, constant vomiting, yellowness of the skin, weak pulse, cramps, and coldness of the extremities.' No looseness of bowels. In a patient who died on the third day, there were evidences of low peritonitis. The spleen was much enlarged and softened, was full of dark blood, and presented, on its under surface, a large gangrenous spot.³

We have few older or more general observations in medicine than that blood dyscrasia and spleen disease have always been present in all severe types of fever: the more malignant the fever, the greater the dyscrasia and the more affected the spleen. It was shown, long ago, by Thomas Wilkinson King, that where, from any cause, blood is prevented from passing readily out of the abdominal veins, the spleen acts as a reservoir, moderating the venous congestion. In Twining's time, and subsequently, much was said about internal congestion leading to inflammation and interstitial deposits, especially abdominal, in grave Indian fevers:—congestion of the veins of the mesentery causing inflammatory effusion between its layers and intestinal hæmorrhage; and again congestion, eventually tending to inflammation of the spleen and liver and to permanent dilatation of the portal vessels. It was the existence of such states, or the belief that they existed, which sent fever-broken, anæmic, sallow, emaciated old Indians, who had just escaped death by bleeding and calomel abroad, to go through a still more trying ordeal of drastic purgative treatment in the chilly, foggy climate of Cheltenham, and which led a

¹ *Ibid.*, January 1, 1880.

² For August 19, 1882, p. 261.

³ Dr. Magaziner Kelinert's Report, from *Russian Military Medical Gazette*, No. 3, 1833.

great Indian physician, thirteen years ago, to say to me, in speaking of our means of obviating the congestive effects of English cold upon the damaged viscera of old Indians—‘Emulge! emulge! emulge!’ I do not deny that, as Indian fevers now are, and as Indian treatment now stands, something of such congestion of the viscera, thus resulting, is observable, but there is not much of it. Splenic disease is, as we have seen, common, especially among the native poor, and there is some hepatic disease, demonstrably of malarious origin; but I am rather content to remain ignorant of the precise causation of these lesions of the solid abdominal viscera than to cling to the old plausible but unproved theory that they resulted from congestion due to arrest of faulty blood in the portal veins, which congestion led to inflammation and enlargement of the spleen and liver. Doubtless Twining was right in considering that, in the grave remittent fever of his day, *unchecked* congestion gave rise to low inflammation and interstitial effusions; but let us ask, What share had the bleeding and salivation in bringing about this stasis of impoverished blood? It must be repeated that, when quinine has fair play, we do not expect to find this congestion.

We see something, but not much, of such passive congestion in neglected cases of remittent among natives. I think that we have two plain facts before us:—

1. Faulty blood causes splenic lesions, as in scurvy, and as shown by the cases of Mutlah fever which I have given, where about five days’ fever, in strong European mariners, just arrived in port, brought the spleen down.

2. Disease of the spleen produces various lesions in the corpuscles, fibrin, and albumen of the blood, *in the causation of which lesions coincident disease of the liver and kidneys probably has a great deal to do*. I have italicised these words because it is certain that, whenever one of the three great solid viscera of the abdomen—spleen, liver, kidneys—is organically diseased, the other two are more or less unhealthy—a fact never to be lost sight of in treatment.

If it were needful to theorise, in the present state of our knowledge, upon the causation of splenic hypertrophy, I would, putting aside the idea of congestive tumefaction, and effusion due to ague and blood dyscrasis, insist upon the close resemblance in many leading points between the process of hypertrophic increment in a *splen ingens* or a leucocythæmic spleen, and the lymphatic mischief which results in scrotal tumour or Cochin leg.

I think we may reasonably look to the helminthologists who have given us an insight into the natural history of *Filaria sanguinis hominis*, and to the demonstrators of *Bacillus anthracis* and *B. malarie*, for inquiries as to whether malarious splenic disease is not characterised by the presence in the affected organ of some animal or vegetable parasite.

It is to be apprehended that those who maintain that paludal fever depends upon the presence of a microphyte in the system assume the existence of that organism wherever such fever prevails. Again, it has not been proved that *filaria sanguinis* is a native of temperate climates; still, even in England, the host of that parasite, the mosquito, makes its appearance in exceptionally hot weather, and its congener, the gnat, is always with us in due season, especially in marshy places. Cats which feed upon cockchafer and black beetles are always ill-conditioned and short-lived. In a part of what was formerly Lambeth Marsh, where my father and I suffered from acute dysentery, we had a *blatta*-devouring cat which died suddenly. I found the bronchial tubes so crammed with *filariæ bronchiales* as to cause surprise that the process of suffocation only occupied a single night.

Sir Joseph Fayrer gives a most important case, in which a young Englishman, returned from India in a state of extreme splenic cachexia and most advanced anæmia, with a spleen descending almost to the pelvis, was becoming much improved under quinine, iron, and good nourishment. 'One day, in spite of earnest warnings to the contrary, he got up, walked to the window, and tried to raise or shut it. He got back to bed exhausted and breathless, and died in a few hours.' Did this death occur from pulmonary embolism, from upward pressure by the enlarged spleen upon the heart, from cardiac weakness, or from inadequate supply of faulty blood to the brain?

Treatment of Splenic Disease.—If there be great pain and tenderness, evidence of splenic peritonitis, we apply sinapisms freely, and keep the bowels gently open, guarding quinine with ipecacuanha, and always bearing in mind that *to relieve spleen, we must remove fever*; and that, *as long as the spleen remains unaffected by treatment, fever is liable to recur, and vice versâ*. The use of frequent doses of purgative medicine was a leading feature in the old showy heroic practice of physic in the East. A course of treatment which caused daily shrinking of the mass recommended itself to everyone. When a patient with recent splenic enlargement happens to get an attack of diarrhœa, this result is often strikingly remarkable.

Considering that Twining pointed out that, in splenic disease, 'intractable diarrhœa appears to be the most common precursor of death in Bengal,' it is strange that purgative spleen mixtures held their ground for some twenty-five years longer, well into my own time—nay, possibly, even up to the present day. Knowing that, in these cases, it is self-evidently our business to nourish and strengthen the patient, and to protect him from all causes of bowel irritation, I always eschewed these evacuant 'spleen mixtures,' giving plenty of quinine and iron, and the best food, maintaining gentle, steady pressure by a broad flannel bandage, and frequently employing sinapisms, and where the case was very obstinate, the galvanic current. Here the great

probability that antiscorbutic treatment is needful must never be overlooked.

A few years ago, I was requested by an old professional brother to see a civil officer, a relative of his, who had returned from the North-Western Provinces of India in a state of splenic cachexia. He had contracted fever and spleen while engaged in his favourite amusement, search for wild fowl—he being an ornithologist, and one of a class, in all times numerous in India, who, not satisfied to wait until fever comes to them, spend much of their leisure in deadly marshes and jungles practically in search for it. I found a middle-aged man, much anæmiated, with a somewhat contracted liver, a *splen ingens*, and a moderate amount of ascites. He had more than once vomited a large quantity of blood. I was told that his urine was albuminous; but either this was a mistake or the condition was transient. I therefore said that we could not use diuretics against the dropsy. He had, not long since, suffered from dysentery. Hence I forbade the use of strong purgatives, knowing how extremely liable these patients are to fatal intestinal flux. Here was what I have been in the habit of calling a ‘constitutional fix.’ Still I said that I thought much could be effected by employing moderate but sustained counter-irritation over the spleen and liver, by bandaging the abdomen, by maintaining normal cutaneous action, and especially by the steady use of quinine. I, however, found everyone’s hand against me. ‘Remove the dropsical effusion by cathartics,’ said a young Indian surgeon; and an old and great London physician (since deceased), who had served for a short time in India, entirely agreed with him. They were clearly of the opinion (which has a very restricted validity in English cases) that drastics are effectual agents in abdominal dropsy—whereas the axiom, *drastics are deadly in Indian dropsies, which, whether we use purgatives or not, commonly terminate by intestinal flux*, is the outcome of my experience. The patient also, oppressed by a sense of abdominal weight, craved for strong purgatives. I and my opinions were, consequently, set aside. I must admit that, as I was informed after his death, he seems to have borne a system of drainage by purgation longer and better than I expected. He lived for many months, being apparently a man of strong constitution; but I believe that he would have recovered had he followed my plan, indicated above, to which, had I found the kidneys healthy, I should have added the use of very moderate diuresis, and a course of ipecacuanha in small doses directed to the liver, the condition of which was, doubtless, one of the causes of the ascites.

The local inunction of an ointment of the biniodide of mercury has the high recommendation of excellent authorities. I may have given it an insufficient trial. The ointment was rubbed over the whole surface corresponding with the enlarged organ, and the native patient was

directed to lie with the abdomen exposed to the sun. The pain was so great, and the raw surface left so extensive, that I abandoned the treatment after having employed it for a short time in a few cases. Dr. Francis Day has seen it salivate in elephantiasis. Long before I went to India, the internal use of mercury in cases of spleen was reprobated by Twining in European practice—unhappily not in that of the native Kobirajes. It produces the most terrible salivation, and often cancrum oris. Those who are the subjects of malarial cachexia are very liable to be salivated by small doses of mercury. When I was inexperienced in the country, I prescribed a grain or two of calomel, in a purgative dose, to a high native official, of unhealthy appearance, who complained of slight hepatic disorder. Severe salivation, promptly checked, and a serious self-inculcated lesson to the prescriber, were the results.

Early in 1852, a middle-aged East Indian, stationed in a very malarious spot near Chittagong, who had several times been under my care for obstinate attacks of intermittent fever, resulting in very considerable enlargement and induration of the spleen, was attacked with epidemic cholera. He was actively treated with mercury by a native doctor. He recovered, but with fearful salivation. I found him greatly reduced, and perfectly blanched by profuse and constant hæmorrhage from the gums, which were sloughing extensively, and exhaling an insupportable fœtor. Every styptic known to the native doctor had been employed in vain. I prescribed an iodine gargle, with nourishing diet and port wine. The hæmorrhage was almost immediately arrested, and recovery was speedy, and as complete as could be expected in one who had suffered so long from malarious influence. It did not appear that the teeth were at all injured.

I had great success in the use of a wash of from two to five drachms of compound tincture of iodine to eight ounces of water, both as a prophylactic and as a cure, in mercurial salivation. Just as the fame of mercury was expiring in India I published the result.¹

Early in 1854, at Howrah, I was called to see a poor European woman, perhaps not of pure blood, who had been attacked with cholera during the preceding night. I had before attended her for hepatic congestion supervening upon chronic dysentery; and she had, for a considerable time previously, suffered from enlargement of the spleen. I found her much collapsed, and the spleen could be felt for a hand's breadth below the ribs. Calomel was then the remedy for cholera, and it certainly appeared to have much to recommend it. Five or six pills containing calomel had already been given by Mr. Burgess. I recommended that, notwithstanding the presence of splenic disease, a grain of calomel should be continued every half-hour. Altogether, about twenty grains were given. The result was favourable; but

¹ *I. A.*, No. 2 for 1854, p. 604.

although a gargle containing three drachms of the tincture of iodine to eight ounces of water was very early employed, moderate salivation set in. The tincture was afterwards increased to five drachms; the mouth remained slightly sore for about a week; at the end of that period, the spleen had become reduced to about one-half of its former size, and she expressed herself as feeling better than she had been for a considerable time previous to the attack. I carefully examined the urine, but could not succeed in detecting the presence of iodine.

Some idea of the prevalence of splenic enlargement among the native poor in the neighbourhood of Calcutta may be formed by driving out upon the main roads in the early morning. Crowds of natives, wearing only the dhotee around their loins, come in tottering under enormous basketsful of vegetables carried on their heads, their spindle-shanks bending æsthetically in all but the right directions. They are active, but generally emaciated, and nearly every one has his epigastrium and his left hypochondrium thickly beset with flat, shiny cicatrices, each of the diameter of a half-crown. These are consequent upon deep eschars produced by the application, generally in childhood, of a hot *gool*, a round mass prepared from charcoal, by which the tobacco is kept alight in hookahs. Certainly this formidable remedy effects many cures. How many deaths it causes is only to be judged of by the not infrequent occurrence of cases like one which was admitted to my ward shortly before I left India—a poor moribund native boy, in extreme splenic cachexia, with a hole completely through the abdominal parietes, caused by sloughing following the application of a *gool*. In like manner, in English practice, blistering over the spleen was often useful, but was found to be very liable to cause sloughing. The natives, considering that a demon has taken up its abode in the spleen, endeavour to kill the intruder by a rude mode of acupuncture, the introduction of iron skewers—H. H. Wilson said, red-hot. I recollect that an ancient rhinoceros, at Barrackpore, was in considerable repute among the natives, who bought and drank his urine as a spleen-cure.

In Calcutta, the Kobirajes frequently give strong sulphuric acid as a cure for spleen. The acid used is generally impure, but it chars whatever it touches. I had four cases at different times. In one, a man took about half a drachm of the plain acid on his tongue. His fauces were whitened. This quantity of fluid, when swallowed, scarcely reaches the stomach. He remained in the hospital for a few days without any bad symptom. More usually, the acid is inclosed in a sweetmeat, capsule-like, and so swallowed. The following occurs among my notes: ‘His spleen was reduced to the volume of a cricket-ball, from having reached the umbilicus. He had taken one drop of *gundhak ke tesab* (sulphuric acid) inside a *buttasha*, or sugar bubble, every morning for a month, which, he said, had cut away the con-

gealed blood in the organ.' A mother and daughter, with spleen, who had been thus treated, were under my care. They were nourished for about six weeks, wholly by enemata. The daughter, but for her cachectic state, might apparently have recovered. In the mother, the corrosive had evidently been propelled by the stomach's action against the pylorus, which was so extremely contracted by firm and even cicatrisation as scarcely to allow the passage of a goose-quill.

When a well-to-do European becomes the subject of splenic enlargement he generally comes to Europe. In some cases, the enlargement, although considerable and very obstinate, refusing to subside under any treatment, appears to give little or no trouble. Not long ago, I watched a brother officer on his way down Regent Street, thinking that England did not contain a more healthy-looking, active, red-blooded man of sixty or upwards. Being an ardent sportsman, he got a huge hard spleen in the jungles of Balasore. This, to my knowledge, remained unreduced for at least twelve years of efficient service in India.

Of late years, removal of enlarged spleens in cases of leukæmia, &c., has been recommended and practised. Thirty-two years ago, I published my opinion ('Medical Jurisprudence for India,' p. 462), that surgeons should be prepared to perform this operation, which I had practised upon the dead body, in traumatic cases; but, even with the record of Credé's and Franzolini's apparently successful cases before us, I am not convinced that we are justified in removing the spleen in any condition of disease.

Spleen Test in Fever.—Chronic splenic hypertrophy is not a sequel of enteric fever in India.

Malarial Hepatic Disease.—Within my bare recollection, enlargement of the liver, following repeated attacks of intermittent, occurred among the denizens of English marshes, who were said to be 'liver-grown.' It is to be trusted that these cases have become fewer since the marsh people in remote districts have supplied themselves with stocks of quinine and Gregory's powder. This state of disease, which, commencing with enlargement, has a tendency to end in kirrrosis, is noticed in nearly all highly malarious districts in India. In large development, it is very far less common in Bengal than splenic hypertrophy, although the liver is rarely, if ever, healthy where the spleen is extremely diseased. It is probable that the ascites, which is common in spleen cases, is largely due to functional or organic hepatic obstruction. I have seen malarious disease of the liver at various stages. It appears to commence as enlargement, which, for a time, diminishes at the end of every attack of fever. Still, every succeeding paroxysm gives increment to the mass. If the fever be completely got rid of, the organ may recover; but, in neglected cases, malarious cachexia is established, and, after many alternations of enlargement and contraction, an extreme degree of kirrrosis is established. The most excessive de-

velopment of the hobnail condition that I ever recollect to have seen—not excepting the gin livers at Guy's Hospital—was in a poor native woman, probably not a drunkard. A medical officer, of very high character, having been stationed at Akyab, a malarious seaport, became the subject of rapidly progressing kirrrosis of the liver. I saw him in London not long before his death from hydrothorax and other dropsical effusions. He was still an active man, between thirty and forty years of age, but with the pinched features and parchment-like complexion often seen in organic hepatic disease.

An officer, who now occupies a high post in India, came to Bengal about thirty-two years ago. I recollect him, on his arrival, a very fine young man, apparently in perfect health. He was sent, early in his career, to a newly occupied station, which was then notoriously malarious, where he had a great deal of fever. In the autumn of 1855, I was asked to see him in Calcutta. He was much wasted and anæmiated, his features pinched, drawn, and painfully anxious, dusky, and pallid. The abdomen was tumid, an enlarged liver extending from high up in the chest nearly to Poupart's ligament, and to the left beyond the navel. To the touch, the mass was of even solidity, generally smooth, except where, below the plane of the umbilicus, a defined hemispherical nodule, as large as half an orange, raised the abdominal walls. I happened to go as far as Madras in the steamer which conveyed him to England. During his first few days on board, the rapidity of his improvement was very striking. His strength and appetite began to return; he was much upon deck; and his liver went up a finger's breadth daily.

I apprehend that, had he lived without change in a very malarious district, the liver would not have recovered, but would, eventually, have become kirrrosed and much contracted. He has always remained very thin, with a pale, shrunk countenance, and has suffered much from neurotic asthma. In these cases, long-continued courses of quinine and ipecacuanha and change of climate are the chief remedies. As mercury is not used in India for hepatic disorder, no caution against the employment of this drug, which would probably kill, is necessary. I have alluded elsewhere to the influence of malarious liver-disease in the production of abdominal dropsy, and I shall have to discuss hereafter the question: 'What influence has malarious cachexia in the causation of hepatic abscess?'

When contraction of the liver causes dropsy, we have before us an important clinical study. As we know that stenosis of one of the ostia of the heart may advance to such a degree as scarcely to allow of the passage of a goose quill before the walls of its chambers lose all power of muscular contraction, so it is almost certain that kirrhotic contraction of the liver very rarely, if ever, goes on to so extreme a degree as absolutely to arrest the portal circulation. At various stages of

confirmed and advancing kirrhotic contraction, the liver becomes congested, say in consequence of an attack of intermittent fever, of a debauch, or of a chill, or of the establishment of one of those states of blood-impoverishment which we designate as 'anæmic.' Now the portal circulation is impeded, and, to relieve this, ascitic effusion occurs. Here is another of what I have termed 'constitutional fixes.' Here we may have emphysematous, bronchitic, or otherwise impedimental lungs, a weak or otherwise obstructive heart, kidneys as faulty as they generally are in cases of hepatic kirrrosis, and a history of hæmatemesis and dysentery. What are we to do here? We can give neither drastics nor diuretics. It remains to us only *to relieve the liver from all removable congestion*. We act here upon the principle which guides us in the treatment of what is called 'spasmodic' stricture, in which there is temporary congestion of a narrowed urethra. We endeavour to avoid tapping, at least until we have fairly tried other means. We give a course of small doses of ipecacuanha, apply large sinapisms frequently over the whole hepatic region and beyond it, keep the bowels regularly open, employ gentle pressure by means of a broad flannel bandage, cut off the drunkard's and generous liver's supply of hydrocarbons, and give quinine in the malarious cases. Should the kidneys be sounder than they are likely to be at an advanced stage of hepatic kirrrosis, we may cautiously promote diuresis. Thus we may effect judiciously what the famous Dr. Sermon did, coarsely and harshly, by use of his 'cathartique and diuretique pill' in General Monk's dropsy.

While his Grace's bowels were sound enough and his kidneys valid enough, they bore and obeyed the evacuants, and the dropsy disappeared. When the hepatic congestion returned, the general health being lower, these coarse evacuants could not be borne, only irritated and failed signally.

We need not fear that, by such a course as I have sketched above, we can, without any strain upon the constitution, at least partially relieve the liver of its congestion; and then, and not till then—(unless the upward pressure of the fluid threatens immediate danger, when we may remove a few quarts through a fine canula)—we may tap with a fair hope of temporary success. I would most earnestly advise the young practitioner never to declare hastily that a kirrrosed liver is hopelessly obstructive. Up to almost the moment of dissolution, the congestions of these contracted livers fluctuate in degree, and are more or less removable. I shall recur to this subject when speaking of anæmic ascites consequent upon malarial cachexia.

Pneumonia.—Doubtless, nine-tenths of the cases of non-epidemic pneumonia which occur are due, not to external circumstances, but to the presence of some constitutional fault. Thus, pneumonia is likely to attack the victims of paludal cachexia, but I have never been able

to trace any relationship more direct than this between malaria and pneumonia. In Lower Bengal—that hot-bed of malaria—I do not think that, in upwards of twenty-seven years, I saw or heard of half a dozen cases of idiopathic pneumonia. I never saw it in a European, but I saw a few cases, and heard of more, in poor, ill-fed natives, in whom the *upper* lobes are especially liable to be attacked.

Dr. Maclean¹ regards pneumonia as the most formidable complication of intermittent fever. He insists that invalids, returning from India or from other hot malarial climates to high latitudes, unless they are carefully protected by suitable clothing, are prone to suffer from this disease. The rapidity with which consolidation of the lungs takes place in such cases is very remarkable. It is not an uncommon thing to see five or six cases of this kind out of one party of invalids landed at Netley from India, if, on entering the Channel, they have been exposed to cold weather. The pneumonia is generally double, and recovery is rare, the patients either sinking at once, or dying after a longer or shorter illness from pneumonic phthisis. So I heard from my father, who was a naval surgeon, that, about the commencement of this century, a Russian squadron having run in to Spithead, the commanding officer was so much struck by the neat appearance of our sailors in their thin blue jackets and trousers, that he made his own people leave off their heavy clothing and gave them a light outfit. The weather was warm, but there were fresh breezes. The poor Russians died in great numbers of pneumonia, as if by a pestilence. Here we have a choice of causes between scorbutus and the influence of the Portsea marshes.

It has been frequently noticed that catarrh prevails extensively in outward-bound vessels on nearing the coast of India. I do not know that it is so, but it may be the fact that, in certain localities, malaria and pneumonia exist in the relationship of cause and effect. Thus malarious Rome is notorious for pneumonia; but, there, the frequency of marked atmospheric transitions may be at least equally to blame. Mr. Oldham has described the complication of intermittent and remittent fevers with pneumonia and pleurisy as very frequent and fatal on the border of the great Indian Desert, as at Bhawalpore. This fact becomes clearer when he adds that, in February 1872, the maximum daily reading in the sun was 107° Fahr., and the average nightly minimum 30° Fahr., giving an average variation of 77° in the twenty-four hours. Still, the question remains: 'Seeing that Calcutta and Chittagong are impested with malaria, and that both places are liable to sudden and very great changes of temperature, why are those places singularly free from pneumonia of strictly pronounced type, if malaria is to be looked upon as a principal cause of that disease?' There is, however, one fact which deserves further consideration and

¹ Quain, p. 734.

examination. I have already noticed the great proclivity of native sick in Bengal to be suddenly attacked with fatal symptoms in the 'small hours.' Here there are generally found what appear to be little more than the evidences of passive congestion of the lungs. Possibly this may be 'pneumonia' which never goes on to consolidation, but kills in three or four hours. To me, however, it appears to be merely that hypostatic pneumonia of the dying which frequently puts a sudden end to grave cases in all climates. The doctrine that, in at least some of its forms, epidemic pneumonia is an acute specific fever, of which the deposit in the air cells is the local expression, nearly as the exanthem is in measles, has been very ably set forth and illustrated¹ by my friend, Dr. Fortescue Fox, of Strathpeffer. On this point, Dr. Gason's report of two cases of intermittent pneumonia, occurring in persons who had been exposed to malaria at Rome, may be consulted with advantage.² I know of two Indian officers who were attacked (one fatally) with pneumonia on visiting Rome. (See also PNEUMONIA under the head of 'Diseases of the Respiratory System.')

I think that, both in India and in Europe, it would be well that attention should be directed to the question: *How far are at least some of the deadly types of bronchial catarrh due to malarial influence?* From time to time, in India, we hear of cases of *capillary bronchitis* or *acute pulmonary oedema*:—A medical officer; a European lady lately married; the young wife of a wealthy Calcutta native, attacked in the chilly early morning with difficulty in breathing, and dying suffocated in a few hours. In the fatal second week of March 1886, when a steady east wind was flooding London with malaria from the marshes of Essex and Kent, an elderly person, who had suffered greatly from the effects of marsh fever in Bengal, got what, at first, appeared to be very moderate feverish catarrh. On the third night, however, the wheezing and cough were so incessant as to suggest the idea that this was a paroxysm of masked intermittent fever in which the sweating stage was represented by a bronchial 'defluxion' so profuse as almost to threaten suffocation. There was some fever, and the urine was loaded with urates. Quinine was given freely, and thirty-two grains were taken in the next twenty-four hours. The first dose, of six grains, was taken at mid-day. Sound sleep followed. Late in the afternoon, wheezing and cough were nearly absent. Next morning, the urine was quite clear. Thenceforward, there was merely some chronic bronchorrhœa. I need hardly say that quinine has been recommended in catarrh and in pneumonia, but not, I believe, upon the indication now suggested. I think that sufficient attention has not been paid to the suggestion of Macculloch that catarrh is imputable to marsh malaria.

Malarial Spasmodic Asthma.—Dr. John Quincy noticed the occur-

¹ *Lancet*, August 29, 1885.

² *M. T.*, vol. i. for 1885.

rence of asthma, as a sequel of intermittent fever, in 1728.¹ Copland² speaks of asthma as appearing as a masked form of intermittent. 'Malaria,' says Dr. Theodore Williams,³ 'plays an important part occasionally in the causation of asthma.' Early in its English history, Bengal became notorious for asthma. It is singular that this fact was the origin of the great Lord Clive's success in life. General Arthur Broome tells us,⁴ that when, in consequence of the events which led to the catastrophe of the 'Black Hole,' a relieving force was ordered to proceed from Madras to Calcutta, in 1756, 'the gallant Colonel Stringer Lawrence, who commanded the Company's force on the coast, was severely afflicted with asthma, to which it was feared that the fog and damps of Bengal would prove so adverse as probably to incapacitate him from active duty. Under these circumstances, Colonel Robert Clive, the second in command of the Company's troops, was selected for this important charge.'

Malarial asthma has been observed elsewhere in India. Dr. Morehead gives the following case—that of a Hindoo drummer, *æt.* thirty, who had suffered for about eight months from intermittent fever and asthma, which was liable to return at intervals of fifteen days. Emphysema was present. The paroxysms of fever and of dyspnoea increased together at night, and ceased towards morning. The asthma and fever ceased in three days, under the use of quinine in four-grain doses, with sulphate of iron and rubefacients to the chest. Dr. Morehead remarks, 'This case is interesting from its bearing on the therapeutic fact that some cases of spasmodic asthma in India are most successfully treated with quinine and small doses of sulphate of iron during the absence of the paroxysm, and in these cases it is reasonable to infer that the asthmatic symptoms have been related to malaria as a cause. To determine the probability of a previous malarious influence, by inquiry into the history of the case and the condition of the spleen, constitutes an important part of the examination of asthmatic patients in India. If there be good reason for suspecting it, quinine and iron are indicated, and a more favourable prognosis than in asthma under other circumstances, provided the emphysema is not great, may be entertained.'

The following is another Bombay case by Dr. Murray:⁵ 'An officer, twelve years in India, who has for the last ten years suffered from asthma, fever, and other diseases, arrived here' (Mahableshtar Convalescent Hill Station) 'in the middle of April, having had ague on the journey; his principal complaint, however, was the difficulty in breathing, which for a long time used to affect him, chiefly at

¹ *Medico-Physical Essays of Agues*, p. 360.

² Dict., part iii. p. 937.

³ Quain, p. 90.

⁴ At p. 77 of his *History of the Rise and Progress of the Bengal Army*, a noble work too little known.

⁵ *I. M. J.*, vol. ix. p. 524.

night (having all the characters of a regular fit of asthma, and rendering him totally unfit for duty on the following day), but latterly these attacks had continued throughout the day. With scarcely any medical treatment, this officer continued almost entirely free from asthma during a two months' residence here; having had only two slight paroxysms during the six weeks preceding his departure.'

In Bengal, I saw many cases in Europeans—five in medical officers of the Bengal Service, and one in a Madras medical officer. I only recollect two in natives—of European habits. Dr. Morehead says that 'it is sufficiently common in natives of India, more particularly in the cold and wet seasons of the year.'

My friend, Dr. Robert Bird, now of Virginia, has recorded¹ the following markedly typical case: 'An engine-driver, at Howrah, near Calcutta, began to suffer from well-marked intermittent fever in the cold weather of 1863, having been previously strong and healthy. These attacks were always complicated with cough. Fever returned again and again, until, at last, the accompanying cough degenerated into chronic bronchitis. But the most interesting feature in this process was that, as the feverish symptoms became less marked, the symptoms of bronchitis became more so, until, at last, the fever entirely disappeared, and the asthma became, for the time, established. After this, he suffered from a fully-developed attack of asthma about once a month, and each attack lasted from two to five days. The disease resisted every kind of treatment, and Dr. Bird had quite despaired of doing the sick man any good, when, in July 1865, he was attacked with fever and ague. From this time, up to March of the following year, when the report was made, he was exempt from the tortures of asthma, with the exception of two very mild attacks.' [I feel confident that removal to a non-malarial district is an absolute condition to complete *permanent* recovery.—N. C.] 'He became perfectly healthy, the bronchitis disappeared, appetite and strength were restored, and he grew stout.'

In nearly all the cases that I have seen or heard of, the disease has been contracted *in paludal situations, within the influence of the sea breeze*. In this respect, it therefore appears to march with elephantoid fever, which has never been known to originate beyond the habitat of the cocoa-nut tree. So much did I mistrust the influence of the sea margin, that about four years ago I endeavoured to persuade a retired brother officer, who after his return to England suffered most distressingly from Bengal asthma, to seek change in a dry spot inland. He, however, went to the Undercliff. He arrived well, but, after a walk over broken ground, had a very severe attack; after this, while I felt that I had not quite sufficient facts to prove that the seaside is not an eligible residence for the subjects of this neurosis, I had

¹ 'Remarks on Malaria and Some of its Effects,' *I. M. G.*, vol. i. p. 56.

the privilege of mentioning my strong suspicion to Dr. E. Headlam Greenhow, who informed me that large experience has convinced him that many asthmatics are made worse by a residence at the sea-side. Dr. Greenhow has known many who, after escaping an attack of spasmodic asthma for long intervals, invariably had a paroxysm on going to the coast, even for a brief visit. This, he added, is not absolutely universal, for he has met with a *very* few exceptional cases. These exceptions may, I think, be due to the law that spasmodic asthma only attacks us *at its own time*. Thus, after I had got over an attack in India, I never feared that any circumstance would again call up the disease for months to come.

Then I have before me the facts that the friend to whose case I have alluded above urged upon me that, when at sea, he has always been free from asthma, and that I, in five voyages to and from India, never experienced the slightest symptom of the malady. I am therefore induced to inquire whether, this disease being essentially paludal, and a large proportion of seaside places, especially in England, having salt marshes in their vicinity, it is not probable that it is *salt marsh* air, not pure *sea* air, which has caused such great affliction to many of us.

Considerable as the bulk of our medical literature on watering places at present is, there appears to be room for a slender tract on the advantages of those few sea-side resorts which are free from marshiness. To seek refreshment in a swamp, at the hottest season, is rather the act of a buffalo or a wild pig than that of a circumspect, well-advised paterfamilias, who desires to afford the blessings of ozone and sea-water to his etiolated flock. Writing on this subject in the 'Medical Times and Gazette' in January 1884, I suggested that it would be well, therefore, to guide public instinct in the right direction; and said that I should be grateful for notes on sea-side places which afford relief to the victims of neurotic asthma, and on British and other European watering-places which are free from marsh. I have not been so fortunate as to receive any reply to this question. I should be sorry to infer from this reticence that every watering-place in Europe has a malarial climate.

I am acquainted with the case of an aged lady, who, residing near land reclaimed from the sea, was asthmatic for many years. Removing to a drier locality on the same coast, she has been wholly relieved.

As I have already mentioned, I became the subject of spasmodic asthma at Chittagong, a very marshy seaside place, after I had begun to suffer from the most tenacious intermittent fever for which that spot is notorious. In the twenty-five cold seasons which I spent in India, I suffered from asthma in twenty-three. I was not constantly asthmatic, but I always expected to be attacked at the setting-in of the cold weather. Of late years, the dyspnoea was not extremely severe, but the attacks were more protracted than formerly, sometimes extend-

ing to six weeks, spasm giving way to bronchitis. In the first twelve years, I believe that I was repeatedly upon the point of death by suffocation. I felt convinced that I was becoming asphyxiated; and that spasm only relaxed when I was beginning to die from exhausted innervation. In ordinary attacks, the dyspnœa was endurable as long as I was able to sit still. Returning to England after eighteen years' service, I spent eighteen months at Cheltenham, where I had a most violent attack of tertian ague and a severe attack of bronchitis, *but not the very slightest symptom of spasmodic asthma*. On returning to Bengal, I again became subject to asthma. In 1872, I came home, and resided for eighteen months in Bayswater, enjoying good health, *and not having a symptom of asthma*, except a vague threatening while staying at a malarious watering-place. The disease recurred on my return to Calcutta. I have now resided in retirement in Bayswater for nearly ten years. I have had more than one severe attack of bronchitis and have experienced threatenings of intermittent fever; but *there has been no sign of asthma*, save an indefinite sense of chest uneasiness once at another watering-place, which, if not built upon a marsh, has extensive marshes on either side of it. I have, for some years, avoided the sea-side. I do not in the least dread the atmosphere of the underground railway, and I do not think that a London fog distresses me more than it does other persons of my age.

In many Indian cases, the amount of spasm is terrible. I was called in the middle of the day to see a youngish man, born, of European parents, in Bengal, in one of my colleague's wards. He had all the appearance of being on the verge of suffocation. His epigastrium was tucked in, almost to his spine, by fixed spasmodic contraction of the diaphragm, and there was so much laryngismus that, for many minutes, I had thoughts of opening his trachea. I afterwards found that he was a very nervous hysterical subject.

In my own and several other cases which I have seen, a few days' asthma leads to profuse bronchorrhœa, attended with great shortness of breath on exertion, which may continue for weeks.

Treatment.—My own case resisted quinine and most other reputed specifics. In the intervals, I took long courses of quinine as a nervine tonic. My brother-officer, whose attacks were markedly periodic, was benefited by quinine. Alcohol was hurtful in my case, and gave me no satisfaction in treating others. When I went to Calcutta, I obtained a fresh stramonium plant, about four feet high and bushy in proportion. As, in course of years, I smoked the whole of this—leaves, fruit, and stem—it probably did me good; but the relief which stramonium smoking gave me was not very considerable. Latterly, I used the English and French cigarettes. Touch-paper and sips of very hot unsweetened coffee gave some little relief. At first, being as much emaciated as an otherwise active man could well be, I starved, re-

stricting my allowance of fluids and solids to the very utmost. Latterly, I took my usual diet, I think with advantage. When ill, all acids, vegetable and mineral, made me worse. I believe that other Bengal asthmatics have been as fortunate as myself in escaping asthma on their return to England. In my own person, I have noticed a peculiarity which I have observed in three other cases—that the right lung is distinctly more troubled than the left, as if the liver and right phrenic nerve were participants in the neurosis.

In my own case, violent and continued morning attacks of *sneezing* with profuse lachrimation, seemed to be an alternative of asthma.

Seeing that my own case covers thirty-five years, I do not think that, even when most obstinate, this neurosis greatly, or at least immediately, endangers the integrity of the heart and air cells. But this, of course, depends much upon constitution and mode of life. The native brother officer, of whom I have spoken, was considered, while in India, to have dilatation of the right heart. He came to England and succumbed more readily than he would probably have done otherwise to an attack of what it is now the fashion to term 'pulmonary congestion,' which I can only recognise as the first stage of double pneumonia.

I am happy to be able to say that my English brother officer, whose case is alluded to above, appears at length to have completely shaken off his malarial asthma; but, some time ago, an attack was excited on passing by rail, a tract of sea marsh between Bournemouth and Southampton, just as terai fever used to be caught in a rapid journey through the belt of jungle at the foot of Indian hills.¹ He now steadily avoids residence by the sea-side. Dr. John Armstrong evidently had a fair understanding of this question when he wrote:—

Skies such as these let every mortal shun

Who dreads

Tertian, corrosive scurvy, moist catarrh,

Nor, for the wealth that all the Indies roll,

Fix near the marshy margin of the main.

Malarial Cardiac Asthenia.—This appropriate designation has been given by Sir Joseph Fayrer² to a state of the heart which is rather frequent among Europeans in Lower Bengal. Recognising the facts that malarious cachexia weakens muscular power generally, and especially tells upon the contractile force of the heart, and that chronic asthma tends to dilate the right heart, it might be supposed that cardiac asthenia would be a frequent attendant of malarial asthma; but this does not generally occur. A. has greatly deranged action of the heart, perhaps with habitual shortness of breath, but without spasmodic asthma; B. has asthma without cardiac distress. In fact, these are

¹ My friend, Dr. Eatwell, tells me that a very delicate lady, advanced in life, has suffered from a return of her Indian intermittent fever whenever she has crossed Pevensey Marsh, near Eastbourne, by rail.

² *Croonian Lectures*, p. 183.

separate neuroses which we do not expect to meet with in the same individual. The ordinary history of a case of cardiac asthenia is as follows :—

A European, who has been long resident in a malarious Indian locality, and who is anæmic, becomes the subject of præcordial uneasiness. This may be very slight, little more than a sense of ‘fluttering,’ or it may be so severe as almost to amount to angina pectoris. In aggravated cases, the patient starts up suddenly, just as he has fallen into his first sleep, with a sense of grave cardiac distress. In advanced cases, the cardiac muscular tissue having become weakened, there is functional dilatation (stretching) of the organ, as evidenced by increased area of præcordial dulness. There is generally shortness of breath in these and other cases of paludal anæmia. An irregular and intermitting pulse is characteristic of this form of cardiac asthenia. Nineteen years ago, I was myself the subject of a temporary disorder which appeared to throw considerable light upon the nature of this condition of the heart. In very pleasant, cool weather, I started with two brother officers from Gowhatty, in Assam, for Shillong in the Kasia Hills. I was in remarkably good health; we had ponies, and the trip was full of interest. After two or three days, my pony broke down, and, none of the others being equal to my weight, I had to walk. I was never in the habit of walking far, and had walked very little for many years. I now walked further, generally up hill, than I had ever done even in my youth. My heart’s action has always been very quiet—almost to weakness. I now found that my pulse was very intermittent. I perceived that this unwonted exercise had stretched the heart’s chambers, and it became a question—is this dilatation to be permanent? This was answered when I found that the pulse improved daily, became regular in less than a week, and remained so. Had I been a smoker, I should have anticipated this occurrence, as tobacco-smoking was evidently a powerful concurrent cause in many of the numerous cases of malarial cardiac asthenia which came under my observation. But, undoubtedly, this form of cardiac debility occurs in ladies, and in men who are not smokers. In all these cases, I gave quinine and iron, and urged return to Europe for many months. In uncomplicated cases, when these measures are adopted early, recovery may, I think, always be predicated. I treated one of my worst cases, an eminent missionary, in 1856. He returned to Scotland in the following year, and has ever since pursued a very active career at home. When I saw him two years ago, he appeared to be a perfectly healthy man of sixty-six.

I never saw *Hay Fever* in India. I lately met an officer who suffered from this malady before he went to India and lost it there. And I have seen more than one person who was violently attacked on returning from India.

Renal Disease.—Albuminuria is often seen among anæmiated Europeans who have frequently suffered from malarial fever in India.

This symptom may attend a condition of apparently vigorous health ; and, as I have found it where the urine was free from casts and had a normal specific gravity, I believe that it may be a mere passive serous hæmorrhage, from spanæmia unattended with permanent organic renal lesion.

Dr. Maclean¹ observes that, in malarial cachexia, 'the urine is sometimes albuminous, with œdema of the lower extremities—symptoms suggestive of Bright's disease, leading to a grave prognosis, often ill-founded, as the above symptoms usually disappear under good climatic and therapeutic means.' I have, however, repeatedly heard of cases of officers of the highest character, who could not be suspected of intemperance, in whom a long Indian career was fatally terminated by what appeared to be Bright's disease. As the patients were not mine, I have no precise knowledge of the conditions of the kidneys in these cases.

Dr. W. J. Moore observes² that 'if the urine passed during or immediately after a fever paroxysm is examined, it will very frequently indeed show evidence of albumen. When fever becomes habitual or of frequent recurrence, albumen is more constantly present. In the more severe forms of malarious cachexy, albuminous urine is quite as often present as absent.'

Sir Andrew Clark has stated³ the noteworthy fact that, of the young men competing for places in the Indian Civil Service examination, he has ascertained, by repeated personal examination, that more than a tenth become albuminuric. He attributes this to 'the strain of prolonged competitive examinations.'

Dr. J. E. Atkinson has given⁴ the results of an inquiry into Bright's disease of malarial origin with the following conclusions. Transitory albuminuria, in the case of fever, is due to intense visceral congestion. It may persist in the intervals. In a proportion of cases, varying with the type of the epidemic, there is inflammation of the kidneys, accompanied by dropsy and the symptoms of nephritis. The usual form of malarial nephritis is the tubal and diffuse variety, the inflammation being apparently most intense in the vicinity of the glomeruli. Contracted kidney may occur as an advanced stage of malarial nephritis, either from long-continued and frequently repeated attacks of malarial fever, or from fibrotic changes such as may ultimately occur in ordinary tubal or diffuse nephritis. It is altogether improbable that this form of malarial renal disease ever occurs primarily, as purely interstitial nephritis. These changes may be induced by any form of malarial fever, though they more commonly follow chronic intermittent fever. The tendency of this lesion is towards recovery ; but, from the persistency of the impaludism or the intensity of the inflammation,

¹ Quain, p. 916.

² *I. A.*, No. 22, p. 281, 'Masked Malarious Fever.'

³ President's Address, Clinical Society of London, 1883.

⁴ *American Journal M. S.*, 1884, and *I. M. G.* for November 1885.

the changes which characterise chronic Bright's disease may be produced. The treatment is to be directed, primarily, to the malarial poisoning, after which the disease should be dealt with as ordinary Bright's disease.

I was lately told by a brother officer, who served in India for twenty-eight years, that, as long ago as 1860, that admirable physician, the late Dr. Edward Goodeve, sent him home from Calcutta dropsical and, it was believed, with confirmed Bright's disease. He has long been a remarkably healthy-looking well-preserved man.

Indian albuminuria might well engage the attention of pathologists in that country, who should begin by examining the urine of a great number of Europeans and natives in apparently good health, then in various diseases, and especially in fever and malarial cachexia. As I have before mentioned, ordinary morbus Brightii occurs in India; but far less frequently than in England. Dr. Morehead devoted a large section of his work to this disease. Many years ago, my friend, Dr. Joseph Ewart, published¹ a very interesting and suggestive series of cases of granular or hobnail kidney in natives, which occurred in the Jail and Dispensary at Ajmeer. The whole subject of renal disease in India would amply repay some years of diligent research.

I think that this inquiry could be best undertaken in those districts of the Madras Presidency and in Ceylon where beriberi is most prevalent. Mr. J. L. Rankin observed, many years ago,² that his experience led him 'to adopt the opinion that beriberi is, primarily and essentially, a renal disease.' I have considered this question under the heading of 'Beriberi,' at p. 68. See *Diseases of the Urinary Organs*.

Blood diseases of malarial origin.—Medical men in the East have reason to be proud of the facts that, of late years, Dr. H. Vandyke Carter has added considerably to our knowledge of the spirillum of relapsing fever,³ and that, Wucherer having detected the filariæ of chylous urine in Bahia in 1866, Dr. T. R. Lewis, A.M.D., now of Netley, made the independent discovery of these nematodes in lymphuria in 1870 (I had an early opportunity of seeing these microzoa). In 1872, Dr. Lewis first observed filariæ, as hæmatozoa, in the blood of a native patient in my hospital. Indian physicians have still, however, a wide field of research open to them in investigating the *bacillus malarie*, and in studying those morbid conditions of the elements of the blood itself which are marked characteristics of malarial cachexia—anæmia, pernicious anæmia, with marrow, &c., lesion (if there be such a disease, apart from the most advanced condition of anæmia, as Dr. Andrew fairly doubts), leucocythæmia, melanæmia, blood disintegration, and consequent deposit of pigment, &c. Up to the present time, these are only known to differ in degree

¹ *I. A.*, No. 4 for 1855, p. 539.

² *Proceedings of the Hyderabad Medical and Physical Society: Report on Beriberi.*

³ *Spirillum Fever . . . as seen in Western India*, 1882.

from the corresponding blood-lesions in Europe. As I remarked, in speaking of the light which has still to be thrown upon the pathology of Indian renal disease, the most promising field for such research appears to lie in those flat and humid districts of Madras, in which beriberi and anasarca are most prevalent. Doubtless, these morbid states of the blood are generally far less developed in Europeans at present than they were during the triumphant career of VS. and mercury in India—when it was a common saying of considerable significance that we ‘went to India green and came back yellow’; and when their generally cadaverous appearance led Theodore Hook to stigmatise old Indians as ‘yellow-bellies.’ Now, the retired Anglo-Indian usually appears to have nearly as large a share of normal red corpuscles as his stay-at-home English neighbours have.

Mr. Hare quotes from Sir Ranald Martin a case of malarial anæmia, which must bring back the remembrance of many like it to old Indian practitioners. An originally plethoric officer, aged thirty-two, who was treated for remittent fever, with bleeding, leeches, calomel to salivation, with a continuous course of powerful purgatives, the diet consisting of sago and arrowroot, is described as being in a state of complete anæmia, with a pale, bloated, lemon-coloured complexion, and mossy, dry, scanty hair; the abdomen doughy and inelastic; the skin dry, harsh, and constricted. ‘This,’ Martin adds, ‘is an example of simple uncomplicated anæmia, resulting from intermittent and remittent fever, and their *necessary* treatment by blood-letting, mercury, purgatives, and low diet. The patient had been under able and experienced medical treatment, but the malarious influences proved too strong to be resisted even by his strong European constitution.’

Hæmorrhages and sanguinolent oozings in malarial cachexia.—

We see, at the instant of the article, as the hue of the visage is changed by the arrest of arterial supply, that thick, mulberry-coloured streams of grumous cruor escape from the nostrils and corner of the mouth. This form of so-called hæmorrhage is the type of those spontaneous oozings which take place in malarial cachexia, which are rarely, if ever, true hæmorrhage, but a voidance of the *débris* of disorganised blood no longer fit for the uses of the system. Wherever there is such dyscrasis of the blood, hæmorrhage from wounds, post-partum, and in menstruation is likely to become formidable.

Although, unquestionably, Indian anæmia was, in old times, largely due to blood-dyscrasis directly resulting from spoliative treatment, marsh poison certainly produces blood disease in that country. Hare, who put down the use of calomel and blood-letting in Indian fevers, writes: ‘In Burmah, at Rangoon, where I witnessed malaria in its most intense forms, I had young healthy patients so poisoned that their blood became as much decomposed as by the poison of a snake-bite, and hæmorrhage took place from the gums and nostrils; and, in

one man with an open bubo, there was uncontrollable dark venous bleeding. These cases were of frequent occurrence, and the face and lips had a blue venous tinge.' I think that, in all probability, there was a scorbutic taint in the above cases. Among the many specious reasons upon which the old practice of blood-letting in fevers was based was the idea of getting rid of bad blood, suggested by these spontaneous oozings of sanguinolent serum, in which, generally, clots do not form, which are, as I believe, nature's own mode of clearing the system of faulty blood. These spontaneous discharges are frequent in India. Sir Ranald Martin told me that, when a young man, being on service in a jungly district, he was attacked by the fever which prevailed in the force, and became insensible. He was awakened from complete unconsciousness, and found himself drenched with blood which had flowed from his nose. Certainly these spontaneous attacks of epistaxis, where the brain has been a good deal affected, appear to do far more good than our leechings do. In the case of an Indian brother officer which I saw after my return to England, violent epistaxis occurred in cholæmia from gall-stone. It was most profuse and obstinate, requiring plugging, but it appeared to be decidedly beneficial, although the sick man was more than seventy years old. I heard, some months ago, of a young gentleman whose health had been much tried in a civil service examination, who, after the ordeal was over, had violent epistaxis which recurred for about eight-and-forty hours. When his son-in-law landed in England, James the Second, being doubtless painfully overworked in mind and body, had several attacks of bleeding from the nose. His death was caused, thirteen years later, by apoplexy.

I was a good deal concerned, many years ago, in the case of a young officer in India who was cashiered for drunkenness. The chief morbid result of the chronic alcoholic poisoning which he had brought upon himself was almost uncontrollable hæmorrhage from the nose and from an ulcer upon the prepuce. I heard, three years ago, of a poor boy, suffering from purpura, who was subject to violent attacks of epistaxis which left him exhausted and anæmiated, but he recovered colour and strength in a few days. A delicate and pale, but healthy girl of twelve, born in India, but taken home in a few months, generally has epistaxis just as a cold in the head is passing off. *Epistaxis* is of very frequent occurrence in India, especially in spleen cases. I have not seen that *periodic epistaxis*, curable by quinine, which has been observed elsewhere.¹ A young and active native subordinate (of European habits) of my own in Calcutta, used, from time to time, to get most formidable hæmorrhage from the bowels. Internal piles were not the cause. He always recovered perfectly in a day or two. It

¹ Copland's Dictionary (Art., 'Intermittent Fever') and *Med. Zeitung*, No. 33, 1836.

must be recollected that here, as in menstruation, Nature elects her own portal—some highly vascular surface, most commonly the nose or intestinal mucous membrane—for collecting and voiding the offending blood-material. She does not call in the surgeon, whose leeches and lancet do not remove the blood when and whence she determines to eliminate it. Consequently, it is not surprising that this clumsy withdrawal of good living blood, of which the system had less than enough, often killed the sick or left them blanched for life. Profuse as these spontaneous ‘hæmorrhages’ often are, we ought, with due regard to the patient’s strength, to be usually disinclined to check them. Almost uncontrollable hæmorrhage from leech-bites was noticed by most of the older Indian writers. Twining says: ‘The altered appearance of the blood in many of these fevers, which arise from exposure to the malaria of the jungles, is very remarkable, and merits careful observation. Connected with this morbid condition of the blood, there appears a disorder of the vascular system which favours prolonged and profuse oozing of blood from leech-bites.’ Hare notices the frequent occurrence of hæmorrhage (most generally in fowls, &c.) after the bite of some poisonous snakes. About three years ago, an old friend asked me what he was to do for dreadful attacks of ague, which commenced soon after his retirement in England. More than thirty years previously, he had a very dangerous attack of jungle remittent at Chittagong. I took no blood from him then. But, upon his getting pain and swelling of one testicle, a frequent sequel of Bengal fever, I applied a few leeches. He felt relieved; but, having gone to sleep after I had attended to the bites, he bled immoderately. Twenty years later, the state of the testicle prevented him from playing cricket. He is now a great pedestrian and lawn tennis player, but probably a chill would bring on a return of Chittagong fever, and perhaps of local pain. In anæmic children, lancing the gums has occasioned serious loss of blood.

Under this head I would place the transitory *Hæmaturia* or *Hæmoglobinuria*, of which I have seen cases in anæmic European children in Lower Bengal. Mr. H. De Tatham gives¹ a case of hæmaturia in a native suffering from scurvy. In the August number of the ‘Practitioner’ for 1868, Dr. Lionel Beale wrote upon ‘Periodic Hæmaturia,’ remarking that the disease is perhaps, after all, more closely allied to ague than to any affection of which hæmaturia is a symptom. The researches of Prof. Murri, of Bologna, appear to bring us still nearer to the conclusion that at least some cases of hæmoglobinuria are of malarial origin. A very full account of this disease, which is now properly designated by Lebert as paroxysmal hæmoglobinuria, as the urine does not contain normal blood-corpuscles, but hæmoglobin, is given by Dr.

¹ *Bombay Medical and Physical Transactions*, 1876, p. 276.

Robert Saundby.¹ Dr. Saundby mentions that there has been a previous history of ague in several of the cases, and that there has been splenic enlargement in some of them, but he considers that these states are exceptional. In its typical form, the attack resembles ague; and it is added that 'a few cases have resided for a length of time in a hot climate.' 'Quinine has seemed of most service.' He holds that 'the relation of this disease to ague is exceptional and not well made out. As the attack is generally excited by exposure to cold, he recommends residence in a tropical climate as a means of warding off attacks. We know that this measure has been tried, but we doubt its prudence. I have seen an attack of this kind (I called it 'hæmaturia' then) on an unusually cold morning in Calcutta; and undoubtedly there are few tropical climates so free from malaria as to invigorate the constitutions of those in whom 'the microscopical characters of the blood are those of slight anæmia.'

The late Dr. Robert Druitt, having been attacked with 'intermittent hæmaturia' in 1866, attributed to a severe chill when he was suffering from overwork, went to Madras in 1873 and 1874, but we are told² that his disease progressed with unequal steps, but always downwards until his death in May 1883. I cannot think the voyages to and fro, and residence in such a climate as that of Madras, at all likely to benefit such a case as this. Hæmoglobinuria is evidently one of nature's modes of clearing the system of faulty blood. Doubtless, medical men in India now distinguish true spontaneous hæmorrhages from discharges of hæmoglobin. My friend Dr. Francis directed attention to this point in 1868.³

At the International Congress of 1881, a paper by Dr. Dreschfield and Mr. Stocks was read, 'On the Hæmoglobinuria produced by Large Doses of Chlorate of Potash,'⁴ and a discussion followed. A woman having taken about an ounce and a half of solid chlorate of potash in twenty-four hours for a slight sore throat, 'was suddenly taken ill with cyanosis and dyspnoea, and passed masses of hæmoglobin both per rectum and per vaginam; the urine and vomited matter also contained masses of hæmoglobin. On the third day, she became slightly jaundiced, while the cyanosis passed off. On the fourth day, she died.' Beyond an enlargement of the spleen, which had been painful to the touch, no organ was found diseased. Either previously existing splenic disease rendered the blood liable to dissolution, or a poisonous dose of potash salt caused the splenic and sanguineous lesions. I have always avoided the use of chlorate of potash in diphtheria, and of large doses of other potash salts in every form of ataxic disease.

¹ *M. T.*, May 1, 1880, and February 4, 1881. *Ibid.*, March 4, 1882.

² *Ibid.*, May 26, 1883.

³ *I. M. G.*, December of that year: 'Periodic Hæmaturia.'

⁴ 'Transactions,' vol. i. p. 398.

Twice in Calcutta, I noticed the occurrence, with an interval of many years, of a type of cholera in which the rice-water stools were tinged with blood. On the first occasion, a considerable number of these cases did so well that I persuaded myself that this moderate loss of blood from the intestinal surface was a good symptom. In the second outbreak, the disease was so fatal that my favourable impression was dispelled. In 1867, Mr. John F. Foster published ¹ the case of an artilleryman at Cawnpore who had suffered during the whole of the previous year from constant but painless diarrhoea, and who was brought to hospital after he had taken part in a brigade parade that morning. At 3 P.M., his stools were very frequent and in large quantity, consisting 'of clear fluid of a deep crimson colour, showing no tendency to coagulate.' There was no straining or tenesmus. The abdomen was hot and very tender. Pulse fast and feeble; tongue coated; head hot, with clammy perspiration upon his brow; countenance extremely anxious; great restlessness and thirst. In an hour, he was collapsed, and purged every ten or twelve minutes. At 9 P.M. the collapse was greater, extremities cold, cramps in arms and legs, abdominal tenderness less, countenance shrunk, voice feeble, and pulse barely perceptible. Constant yawning and extreme restlessness. No vomiting or sensation of sickness. The motions became gradually of a lighter colour, and contained flocculent particles. Collapse increased until his arms and legs became blue; pulse imperceptible at the wrist; faculties unimpaired. He expired with difficult breathing in twenty-two hours from the commencement of the attack. The ascending colon was slightly congested, and many of the solitary glands were surrounded by a thin zone of blood effused beneath the mucous membrane; but there was no ulceration or breach of tissue. Both small and large intestines were distended with clear fluid of a light yellow colour, in which floated numerous flocculent particles like those in the rice-water dejections of cholera. The spleen weighed one pound eight ounces, was slate-coloured, and of very pulpy consistence. This patient had suffered from two attacks of hepatitis and one of splenitis; but not from dysentery, diarrhoea, or scurvy.

Similar as this case was, in some of its leading features, to one of hæmorrhagic cholera, I quite coincide in the opinion of Mr. Foster and the Editor of the 'Gazette' that it was one of remittent fever; I would say a pernicious form of that disease, closely linking it with true cholera. The Editor mentions that similar cases were, he believes, met with in men of the 101st Regiment, who were saturated with malarial poison when that corps was stationed at Mooltan a few years previously. He adds: 'The effect of malaria in inducing a state of the blood favourable to the occurrence of hæmorrhage is too well known at Peshawur.' Two other cases, much resembling Mr. Foster's, of perni-

¹ *I. M. G.* for December of that year.

cious malarial fever with bloody flux, are given by Baboo Ooday Chand Dutt. I have cited them in tracing the close relationship which exists between pernicious remittent fever and cholera. I only notice these cases here on account of their markedly hæmorrhagic character, although the disease which they characterise is no mere hæmorrhage.

In the above article, Mr. Foster mentions that he has notes of a case of fever which came under his observation, which was rapidly fatal in consequence of *passive hæmorrhage from the whole of the mucous membrane of the lungs*—a characteristic occurrence in black death, sweating sickness, Pali plague, and mahamurree.

In a report published in 1842,¹ Mr. Leith notices a case of '*Intermittent Menorrhagia*, of tertian type, in an Indo-Briton (half-caste), wife of a soldier, which was cured with quinine given in repeated doses close on the expected time of return.' I have seen a great deal of very troublesome menorrhagia in Bengal; but, perhaps by blamable oversight, never observed that it assumed an intermittent character. There is generally amenorrhœa in splenic cachexia.

Post-partum Hæmorrhage often due to malarial influence, is probably the greatest and most frequent danger attendant upon parturition in India. I shall revert to this subject in speaking of puerperal fever.

Hæmatemesis is a not very frequent occurrence in splenic disease, but I know of two marked cases in officers. It was observed by Dr. Duncan Stewart that such hæmorrhage 'often effects a salutary change in the diseased organ, and conduces to its cure.' Doubtless, when the hæmorrhage is large, the size of the spleen is temporarily reduced; but, in my experience, hæmatemesis only adds gravely to the sum of splenic cachexia and its attendant anæmia.

Those practising in Bengal have abundant evidence of the fact that the marsh poison and deficient food induce extreme poverty of the blood. Sir Ranald Martin noticed that, in operating at the old Chandney Hospital, Calcutta, the blood often looked like thin claret.² I have repeatedly seen this, especially in a poor fisherman whose thigh I amputated in consequence of a desperate crocodile-bite. After every visible artery had been secured, there was oozing of thin dark blood from the whole surface of the stump until he sank.

With these expressions of states of blood-disintegration must be ranged the bloody sweats of plague and malignant fever described by Hodges and Huxham;³ the 'hæmorrhages,' ecchymoses, and cardiac

¹ *I. M. J.*, vol. ix. p. 710.

² Huxham says, in speaking of bloody sweat: 'It is observable that, when this sort of hæmorrhage happens from the nose, the matter is a thin bloody ichor, not concreting as blood commonly doth from the nose of persons in health, or in an inflammatory fever.' 'Some chlorotic girls are vastly apt to bleed from the nose, and yet their blood doth but just colour a linen cloth.'

³ Huxham had a very fair idea of embolism and pigment plugging. 'The *petechiæ*, *vibices*, or livid *stigmata* that very often attend these hæmorrhages, show

blood-concretions of scorbutus ; and also the yellow suffusions, petechiæ, 'flea-bitis,' and black vomit of yellow fever, of the relapsing fever of India, of the yellow plague of the middle ages, and of certain grave forms of tropical paludal remittent.

The whole of the Indian blood-diseases ought to be carefully re-investigated in that country—a multitude of points, such as the following, being thoroughly wrought out in localities where malarial cachexia is most prevalent and of most extreme intensity, in both Europeans and natives. Hertz's observations on blood pigmentation in anæmia should, as Sir Joseph Fayrer indicates, be practically followed out. Dr. Gowers found that 4 per cent. of cases of lymphadenoma were related to intermittent fever. This disease occurred—but very rarely, in my Indian practice. The condition of the blood and viscera should be examined as far as possible in all such cases. Twenty-five per cent. only of Dr. Gowers' cases of splenic leucocythæmia were found to have either suffered from intermittent, or to have lived in an ague district. A complete series of observations upon the red and white corpuscles in Indian malarial cachexia is needful. What of progressive pernicious anæmia in pregnancy (which is not considered to be malarious) in India? What also of *leukæmia-myelogenica* in that country?

In the majority of cases, malarial anæmia is distinguishable by a single glance at the patient's countenance; but in some cases, especially in young women, the roundness (in reality, œdematous puffiness) of the face is, just at first sight, deceptive. In naturally florid Europeans, the colour of the lips and cheeks is sometimes misleading; but, in all severe cases, the blanched conjunctiva of the lower lid is distinctive. Daily examination of the eyelid is our guide in the employment of quinine, iron, and nourishing diet.

Among the *diseased conditions consequent upon malarial cachexia* are œdema, ascites, and hydrocele. Each of these demands separate notice.

Œdema.—More or less puffiness of the face, ankles, and sometimes of the hands, is characteristic of severe Indian malarial anæmia. Some œdema of the ankles is very frequently seen among old Indians. I (in common, I believe, with many other medical men) long regarded this condition of the ankles as being, in all cases, a very grave evidence of a broken constitution. Dr. John Radcliff's brutal remark to William the Third—'I would not take your Majesty's two legs for your three kingdoms!'—has, doubtless, ever since rankled in many minds. I, for one, did not give sufficient weight to the common facts that unseasoned female nurses and shopwomen, and sailors who have become a little scorbutic on long voyages, get œdema of the lower extremities; and that there is always more or less swelling of the feet, suggesting that the blood-globules are dissolved, or broken down, and enter into the serous arteries, *vasa exhalentia*, &c., where, sticking fast, they form these appearances.'

slippers, after a long walk. We all know what is the true significance of this symptom in fixed organic disease, cardiac, pulmonary, renal, &c.; and, in hospital practice, the student, observing the almost constant attendance of this symptom upon deadly chronic maladies, naturally imagines that it is always a harbinger of death. A sensible writer has said that a large proportion of our worst hospital cases only seek admission when œdema has become so great as *to prevent them from wearing their boots*; otherwise, they would go about their business until they sank from debility. Men in private practice, in England and in India, see a great deal of œdema of the ankles, unattended with serious organic disease. Many ruddy and active old Indians are subject to this condition. Some time ago, I was making a few calls among my Indian friends, and saw a brother officer, in whom œdema of the lower limbs was really a symptom of advanced organic disease. In thinking over his case, I recollected that I had chanced to see, on that day, six fairly healthy people from India, whom I knew to be then, or to have been at some time or other, subject to this condition. Doubtless this œdema arises from a variety of causes in different subjects; and it is probably a graver symptom in some cases than in others. In all, anæmia appears to be present while the œdema lasts, and is evidently diminished when that symptom disappears. Considering that I know of persons now living, in by no means very weak health, who became subject to œdema of the lower limbs in India, fourteen, sixteen, and twenty years ago, it is evident that this condition is not always attended with great danger. In 1888, I saw the report of the death of a senior brother officer, the statement of whose case I, as Secretary to the Medical Board, read in 1857. He had undergone considerable hardship in the siege of Saugor, was probably rather scorbutic, and was described as being dropsical and œdematous. He subsequently resided at Cheltenham for a quarter of a century, dying at the age of seventy-eight.

Ascites,¹ quite independent of any discoverable disease of other organs, is very common among natives of India suffering from enlarged

¹ The following is a good typical case of this anæmial œdema, occurring in a very old Indian. In a letter, written from Daylesford in May 1809, ten years before his death, and when he was seventy-six years old, Warren Hastings said: 'My own case has something of novelty in it. I left Portland Street a very invalid: I arrived almost well at Tettsworth, and the next day in perfect health at Daylesford, though my nights were infested with feverish returns. These, however, being perceptibly abating, I am now, and have been for some time past, in much better health than I had before my late indisposition; yet, as I grew well, a new disease succeeded—a swelling in both my legs and feet. This increased to such a degree in my right foot as to be alarming, and its appearance to my sight disgusting. By Dr. Vaughan's prescription I have taken for it sweet spirit of nitre diluted with imperial three times a day; but I am not sensible of any amendment. Any exertion aggravates the affection of the part, and the flesh-brush instantly allays it.' In another letter,

spleen and malarial cachexia. At first, I tapped some of these poor creatures,¹ but I had very bad success in most of my cases of paracentesis; and latterly, for several years, found that, by treating the cachexia and by carefully bandaging the abdomen, nearly all cases of merely anæmic ascites, not admitted moribund, did well, the fluid disappearing more or less readily without the use of diuretics, cathartics, or other evacnants.

Unquestionably, hepatic obstruction, temporary or permanent, generally has much to do with the causation of ascites in spleen cases. I have met with cases which afforded me assurance that, when the system is lowered by paludal cachexia, a state of liver disease which is temporarily obstructive to the circulation, but which has not reached the condition of permanent kirrrosis, may lead to abdominal dropsy, which may be relieved by change of climate, by steady counter-irritation over the hepatic region, and by a course of ipecacuanha in small doses, combined with all those means by which we treat malarial anæmia. It is needful to examine the state of the liver most carefully in all these cases.

Cardiac Anæmia.—Here it may be noticed that a deteriorated condition of the blood is more operative than mechanical obstruction is in the causation of dropsy in 'obstructive' disease of the heart and liver; thus:

A. and B. suffer, at the same age, from the same degree of rheumatic stenosis of the aortic orifice.

A. lives for many years, and dies, probably suddenly, without dropsy.

B. dies in a few years with chronic dropsy. Here many causes of death are at work, but the main causation is that:

apparently written in the same year, he says, rather confusedly: 'The swelling of my legs has subsided, and come almost to their bearings, taking the word in every sense. This effect has been produced neither by pills, by galvanism, by repose, nor by diet, but by, or in despite of, severe exercise of them, and evidently more than is good for them at the time that I impose it upon them.'—Warren Hastings in Slippers—*Calcutta Review* for March 1856. I am inclined to think that this very chronic œdema, when it occurs in old Indians, is allied to elephantiasis.

¹ In many cases, they are not brought to us until the distension is so extreme that we are compelled to relieve the upward pressure of the diaphragm upon the lungs by drawing off fluid. I asked one poor woman, on admission, how long she had suffered from dropsy. She replied, '*Two years.*' She was so weak that, although the distension threatened suffocation, she was obliged to lie without a pillow, evidently that sufficient blood might reach the brain. I asked my colleague, Dr. Edward Goodeve, who was passing my ward, to look at her, saying that I hated tapping, and knew that, if I tapped her, she would certainly die; and that, if I did not, she must inevitably die. 'Then,' said my friend, 'give her the benefit of going out of the world *secundum artem*,' and he lent me a very delicate trocar, with which I let out only sufficient fluid to relieve the diaphragm. She sank in two days.

A. lives in such a manner that his blood does not become deteriorated, breathes good air, avoids fatigue, has the best food, and only dies when arctation of the aortic ostium, and consequent dilatation of the left ventricle, have proceeded to the utmost.

B. is obliged to work hard in a malarious place, has bad food, is ill-clad; he, being inadequately nourished, becomes in a degree scorbutic or anæmic; the life of his blood is deteriorated, the muscular power of the left ventricle rapidly fails, and dropsy occurs under a very moderate amount of cardiac obstruction. Consequently, one of the leading therapeutic indications in these cases is to keep the blood in good condition.

Hydrocele is a disease of very great prevalence among the natives of Lower Bengal, chiefly the poor, but many of the rich suffering. Sir Joseph Fayrer has published¹ an important paper in which he holds that this 'is one of the many forms in which the influence called "malaria" expresses itself.' This is undoubtedly the case. As Secretary to the Medical Board, I saw nearly all the officers who were sent down to Calcutta sick during the Great Mutiny of '57; many who had undergone great fatigue, exposure, and privation, especially during the siege of Lucknow, suffered from hydrocele, which I attributed, in part, to scorbutus.

[*Vide* 'Diseases of the Male Organs of Generation.']

In malarial blood-disease, various *embolic conditions*, such as vessel-plugging, phlegmasia dolens, and gangrene of the extremities, are very liable to occur. Both arterial and venous embolism are not infrequently observed as complications of paludal cachexia in temperate climates; these lesions are probably more frequent in India.

Venous Plugging.—Dr. J. Mouat, H.M. 13th Dragoons, published² the cases of two dragoons suffering respectively from malarial fever and from dysentery, who were both attacked, while under treatment in the notoriously unhealthy hospital at Bangalore, with extensive plugging of the veins of the lower limbs, as in phlegmasia dolens, and who appear to have died from pulmonary embolism.

Mr. Greig records³ the case of a European who, having been bled for continued fever, died from phlebitis. In his remarks⁴ upon the case of a European soldier, suffering from dysentery, who died of phlebitis consequent upon VS., Dr. Macgregor says: 'During the latter part of July, scarcely a man was bled without the risk of producing inflammation of the vein.' He appears to attribute this to the impurity of the hospital air.⁵

¹ *Clinical and Pathological Observations in India*, p. 513.

² *C. M. T.*, vol. viii. part i. p. 28.

³ *Ibid.*, vol. v. p. 101.

⁴ *Op. cit.*, Appendix, p. cxiv.

⁵ During his eleven days in hospital, this poor man lost two pounds of blood by VS. twice, and was again bled to syncope, and had twenty leeches applied in the

At Chittagong, a portion of one of my external thoracic veins became plugged, without pain or any sign of active inflammation. An old man, one of my table servants, came to me with a corded state of the saphena vein at the middle of the thigh. An abscess formed externally and was opened. There were no other bad symptoms.

Phlegmasia dolens after delivery is a rather frequent occurrence in women who have suffered from malarial fever in Bengal.

Arterial Plugging.—When a student at Guy's, I had the case of an anæmiated lad, one of whose hands went quite spontaneously into a state of dry gangrene from arterial plugging. Dr. Carter narrates the case¹ of a Mussulman beggar of Bombay, aged thirty-two, who was an opium-eater, and who had suffered from great exposure in stormy weather, who died from gangrene of the feet consequent upon plugging of the abdominal aorta. [Prep. C1 in the Catalogue of the Bombay Collection shows 'plugging of the abdominal aorta' at the point where the common iliacs arise.] Sir Joseph Fayrer has given a very interesting set of cases of natives suffering from gangrene of the extremities caused by arterial plugging of distinctly malarial origin. In one year, I saw more of these cases at the Calcutta Medical College Hospital than I did in all the rest of my Indian experience. There was evidently something peculiar in the endemic constitution of that year. At this time, I saw, with my friend Dr. Robert Bird, of Howrah, a Jewish girl of seventeen, the child of rich parents, whom I had attended before for congenital heart-disease, dying from arterial gangrene of both lower limbs. Some years previously, I was consulted in Calcutta in the case of a European uncovenanted judge, whose femoral became plugged high up, with the remarkable and most painful result of causing sloughing of the whole integument of the lower extremity, producing an enormous expanse of ulcer which refused to heal. Sir Joseph Fayrer notices that anthrax and boils and abscesses, so common in India, are attributed to capillary obstruction by pigment—the *débris* of broken-down blood-corpuscles; he would rather attribute them and other consequences of obstruction to fibrinosis. During a long course of years, Sir J. Fayrer observed, in India, the occurrence of ante-mortem coagula in the right heart as a cause of death after surgical operations and accidental injuries, in cholera, and in various forms of malarial cachexia. The result is communicated at page 94 of his 'Clinical and Pathological Observations.' Hertz² describes gangrene in pernicious fever, affecting the leg and the female genitals

course of the inflamed vein. On post-mortem examination, 'there was only a slight blush in the colon, showing that active inflammation had existed and was subdued by the repeated use of the lancet.' In the present day, one large dose of ipecacuanha would probably have saved all this trouble.

¹ *Bombay Medical and Physical Transactions* for 1860.

² In Ziemssen.

[was not this malarious phagedæna?]. A girl's hands, first one and then the other, became gangrenous after a few days of intermittent fever.¹

Sloughing of the Cornea and spontaneous gangrene of the Scrotum and Perineum are, in some years, frequent sequelæ of malarious fever and cholera among the ill-fed poor of Bengal. This mischief, occurring in the perineum, has not, as far as I am aware, been commented upon as occurring elsewhere. Most of the cases which I saw were in native prisoners. One's attention is called by the native doctor to a man with extravasation of urine, but there is no evidence that sudden rupture of the urethra has occurred. Upon making free incision, it is found that the whole perineum is occupied by a huge gangrenous abscess, which in some cases does, and in others does not, communicate with the urethra. In these cases, I used to say that the man had begun to die at the perineum. A distinguished officer died in this manner in Bengal some years ago. I believe that, elsewhere, extravasation of urine, in old stricture cases, generally results from the formation of abscess outside that portion of the urethra which gives way. When at Howrah, I was called to see an elderly native who was famed as the wealthiest man in Calcutta. He had, after suffering from fever, begun to die at the scrotum, and expired in a few hours after my visit. As far as I am aware, these forms of gangrene of the perineum and scrotum are always speedily fatal. Many of those who suffer from *sloughing of the corneæ* (which, in its causation, appears to resemble that sloughing of the corneæ which Magendie produced in dogs by feeding them exclusively on bread) sink—death commencing at the corneæ. But, in a long course of years, a good many of my patients recovered. I, at first, adopted the treatment much employed by my friend Mr. Samuel Browne, of Belfast—the application of a saturated solution of nitrate of silver. With this, I obtained some success. But, latterly, at the recommendation of Mr. C. Macnamara, I merely closed the eyes with fine cotton wadding, due attention being paid to cleanliness. Under this treatment, a considerable proportion of cases recovered, especially when they were taken early, and when only one eye was affected. We always looked for this sloughing in every low case. The first sign is a generally sunken appearance of the cornea, such as is seen after death, with a cloud of

¹ Dry gangrene, due to other than a malarial cause, may, of course, occur in India. Dr. Campbell saw a case (*Bengal Medical and Physical Transactions*, vol. i. p. 287) in which it may have been due to eating diseased grain. My friend Dr. Theodore Duka records the case of a young Madrassee woman, aged fifteen years and a half, in the eighth month of her first pregnancy, which occurred at Simla. There was dry gangrene of both legs to within three inches of the knee-joints. Amputation was performed; no bleeding vessel had to be secured. The patient had an easy recovery, and gave birth to a well-formed full-grown child. No enlargement of the spleen or liver could be detected.

milky opacity of a segment of its lower edge, as if its attachment to the sclerotic had been slightly separated with a cornea knife. Of course, quinine, wine, and the best nourishment are indispensable in these cases.

Malarial Phagedæna (cancrum oris), although occurring in the same class of cases of malarial cachexia, especially in advanced splenic disease, is not attributable to the same immediate cause as the gangrenes above described are, the plugging of afferent or efferent bloodvessels. It is not simple gangrene, but is a true sloughing phagedæna. In many of these cases, the patients are brought to us death-stricken, and expire in a few hours without separation of the slough. This looks like simple gangrene, but it is not. When we can get the slough to separate, leaving a clean surface, we notice that, while the ulcer may be healing at two-thirds of its circumference, the edge of the remaining segment is being eaten away by phagedænic ulceration, and that the sore is rapidly extending at that part. I have seen *cancrum oris* (only in young children) in England, before I went to India and since my retirement. The disease appears to be nearly the same in this country and in India. There, in old times, it used frequently to be caused by the use of mercury in our hospitals. Three grains of calomel would set it up in a cachectic subject. Now, cases are brought to us where it is attributable to mercury given by Kobirajes. But we often observe its appearance in our own wards in patients who have not taken mercury. It generally occurs in cases of splenic cachexia, after repeated attacks of malarial fever. Soon after I joined, I was surprised to see the disease in a middle-aged native man in my jail hospital at Chittagong. In 1834, the editor of the 'Indian Journal of Medical Science' mentioned the case of a young sepoy who had taken five grains of calomel in two pills on account of a slight attack of fever. In about thirty hours, his face began to swell. There was no salivation; the left cheek 'completely sphacelated away, and the poor fellow sank.' Dr. Macgregor gives¹ the fatal case of a European soldier, *ætat.* 21, who had suffered from dysentery, dropsy, and fever; and a second, the report of whose case is headed, 'Dysentery and Dry Gangrene succeeding the accumulation of Calomel in the system.' A case occurring in a European soldier, *ætat.* 22, with enlarged spleen, got at Roorkee, has been published by Mr. P. W. O'Gorman.² In 1836, Dr. Corbyn mentioned³ that, some years previously, in Arracan, he had seen a great deal of phagedænic affections which resisted every application employed, although many of these had proved efficacious in checking similar disease elsewhere. Phagedæna invariably supervened when the constitution had been previously debilitated by repeated attacks of the Arracan remittent fever, in which disease the

¹ *Op. cit.*, pp. ix. and lxii.

² *I. M. G.* for January 1882.

³ *I. M. J.*, vol. iii. p. 545,

spleen was always more or less affected. A great number of patients were sent to a hospital formed at Barrackpore for their reception, but the failure of every means adopted was the lamentable result. I think that, upon reviewing the history of this disease, in cases not attributable to mercurialisation, and of venereal *noma pudendi* of the very poorest girls, and of 'black lion' in soldiers in trying campaigns, and in sailors just come in from long voyages, it will be found that *scorbutus* is almost always a factor in the causation of sloughing phagedæna. Three forms of this disease are common, in paludal cases, in Bengal. They commence as follows:—either (1) as a swelling of the cheek, with a slight vascular blush, in the European, commencing on one side at the commissure of the lips, and going on to commit almost unlimited destruction of soft parts and bone. This form has always been fatal in my observation; although, in a few cases, the progress of ulceration has been tardy. (2) On turning down the lower lip, a sloughing ulcer is seen in the mesian line where the mucous membrane of the lip is reflected upon the gum of the incisors. Here ulceration may go on to destroy the lip, alveolus, and more. (3) Opening the mouth widely, a portion of the ascending ramus of the lower jaw, about the size of a finger-nail, is seen, white like a piece of tooth-brush handle, and quite denuded of mucous membrane and periosteum. When the second and third lesions occurred in patients under treatment in my beds, and I was able to act early, I now and then succeeded in bringing them through by means of quinine, support, and the daily application of the compound tincture of benzoin to the diseased surface. Some used nitric acid; but, where there was so little vitality, I preferred the above stimulant.

In 1836, Mr. Egerton described¹ an outbreak of *noma pudendi* which had lately occurred in the Lower Orphan School of Calcutta, which was crowded. Children came in with fever; head hot, countenance sunk. They continued in a state of low fever from ten to fifteen or twenty days; when, in one case especially, sloughing supervened; an ulcer first appeared on the clitoris, spreading to the mons veneris and labia, which sloughed off. The girl recovered. Preparation 549 in the Calcutta Medical College Museum is 'enlargement of the spleen, taken from a girl who had been a sufferer from sloughing ulceration of the labia and the soft parts over the pubes.'

In some cases, especially when we can give quinine and support early, the ulceration becomes chronic. I had a native lad with large spleen, in whom the disease assumed the first form, commencing at the cheek. After great part of the cheek had come away, what would, in a dog, have been one of the intermaxillary bones, separated, and was drawn out, and was made a preparation in our museum. The whole of the inner portion of the upper jaw, with two incisors and a

¹ *Ibid.* vol. iii. p. 545.

canine tooth, and the palatine and nasal processes, are there. He improved in health; took his food, wine, and quinine well; the raw edges being quite clean. He kept a little hand-glass, in which he used to examine his face; and we became so hopeful that we might save him, that I consulted my friend Dr. Phin Smith, who thought that he could supply the loss by an apparatus of silver and gutta-percha. At length, the whole of the integumentary edge would appear to be cicatrised, but, upon carefully examining this all round, it would be found that, somewhere or other, ulceration was undermining the margin and eating it away, as a spark extends on touch-paper. He died after he had been with me for several weeks.

At Chittagong, my assistant Baboo Ramkinoo Dutt showed me a native boy with spleen, in whom the application, by a Kobiraj, of a gool, as an issue, had caused destruction of all the soft parts, integument, and extensor muscles of the whole of the back of one forearm, so that the shafts of the radius and ulna lay exposed throughout their course from elbow to wrist. Most careful dressing was being rewarded by the appearance of a clean surface and a few granulations.

An important development of malarious cachexia is *Neurotic Neuralgia*.

Malarial Neuralgia, in its various forms—*facial* and *hemicranial*, *brow* *ague* and *intermittent headache*—and neuralgic affections of other nerves, as the *sciatic*, *spermatic*,¹ &c., are, with the exception of the last, much severer and far more prevalent results of malarial poisoning in England than they are in India. Although, in that country, we see neuralgia march side by side with paludal fever, this neurosis appears to be the special congener of those fevers of which strong *ague* is a marked characteristic.

Two singular forms of malarial neuralgia are, however, frequent in our Eastern possessions—*Burning of the Feet* and *Neuralgia of the Shins*.

The former affection was the subject of monographs by Drs. Grierson,² Malcolmson,³ McKenna,⁴ and Waring.⁵

It is, according to Waring, most prevalent in Arracan, Burmah, the Tenasserim Provinces, Penang, Singapore, and our other Eastern Settlements, less so on the continent of India, although it occurs there in the hospitals of native regiments. I never saw or heard of but one case in Lower Bengal. The patient was a fine-looking but rather thin sepoy, who was sent from Fort William to be placed in my ward for observation. A high official came, inspected him with me, and asked my opinion. The man sat on his bed with his feet on a pillow, the soles being turned inward, so as to face one another for

¹ Here pain usually depends on local organic changes, as in elephantoid disease.

² *C. M. T.*, vol. ii. for 1826, p. 275.

³ *Work on Beriberi*, part 2, p. 40.

⁴ *Madras Quart. Med. Jour.*, vol. i. p. 432.

⁵ *Ibid.* vol. i. 1860, p. 300.

protection. The attacks were paroxysmal, and the soles could not even bear the contact of a sheet. I said that I considered it to be a case of burning of the feet, as described by Malcolmson. The reply was, 'I never heard of Malcolmson, or of burning of the feet; I think that he is malingering!' A young medical officer, recently from Netley, who stood by, said, 'I have read Malcolmson on burning of the feet.' I added, 'He is certainly not malingering; look at his agonised features and the beads of sweat on his forehead.' I employed quinine and all other feasible means, but could do little for the poor fellow's relief. Grierson and Waring attribute this disease mainly to malarial influence. Mr. Playfair¹ describes two forms of this disease as being commonly met with, both in the feet and hands: (1) in which the parts affected are in a constant state of moisture, from sudor; (2) the other, in which the extremities are dry, and sometimes scaly; but there are several varieties. In some, the feet only are affected; in others, the hands; in many, both hands and feet. In a few, the feet are dry, while the hands are bathed in sweat; but, in all, the disease is very distressing, generally preventing all natural rest, and disordering the animal functions in a remarkable degree. The patients describe their sufferings as nearly insupportable. Waring remarks upon the difficulty which has always been experienced in curing this disease. He considers that, as dyspepsia, with light clay-coloured yeasty stools, appears to exist in the majority of cases, our first endeavour should be directed towards establishing a healthy condition of the assimilative functions. Dr. McKenna relied much upon the liquor arsenicalis, in four-drop doses, and iron and quinine, where the exacerbations are distinct. As local applications, the leaves of *Mhindi* (*Lawsonia alba*), strong solutions of opium and iodine, and aconitine ointment, are recommended by Waring, who, however, insists upon early removal of the patient to his native place. The detention of sepoys in the Eastern settlements often proved fatal.

Many ill-fed Bengalees suffer greatly from *Neuralgic Pains in the Shins*, which has been recognised as a disorder allied to burning of the feet. The patient sits, with a disconsolate aspect, on his bed, his knees bent up, and his feet flat and drawn under him, 'hunkering' in the usual native manner; but he holds his hands protectingly on either side of his shins, with the fingers turned inwards, suggesting the notion that his tibiæ are musical instruments, the keys of which he would touch delicately, if they were not too sensitive. At my first station, one of my earliest patients was a prisoner in the jail hospital thus afflicted. Considering that the pain was rheumatic, I gave vinum colchici. In a short time, diarrhœa set in, then both corneæ sloughed. My compunction was extreme; and I fear that my sense of relief amounted to satisfaction, when, in a day or two, other patients, who

¹ C. M. T., vol. ii. p. 280.

had not taken colchicum, got sloughing of the corneæ. Gout not being prevalent in India, I never prescribed colchicum again.

Dr. Morehead has a very interesting note (page 654) on *Paroxysmal Headache*. Cases of *Hemicrania*, *Intermittent Cephalalgia*, and moderate *Brow Aque*, generally yield, without any great obstinacy, to quinine in Bengal.

I have not seen a great deal of *Migraine*; but, in Bengal, especially among men who overwork their brains, it is well marked, and is inclined to display periodicity. In one case, it came every Thursday—this was not my case—but quinine did not appear to touch it. I saw cases of *Facial Neuralgia* distinctly marked, but few. My father, a naval surgeon, was, I believe, one of the first, probably the very first, who recommended large doses of quinine in facial neuralgia; and I consider it due to his memory to give a brief record of the facts. About the year 1810, he had charge of a French prison hospital ship moored near a salt marsh at Plymouth. His duty occasioned him extreme anxiety and vexation, and there he had his first attack of *tic*. His second attack was at Greenhithe, in the Kentish marshes, in 1823. Although I was then a very young child, I have a vivid recollection of the strong man's almost intolerable agony. With desperate firmness he swallowed large quantities of cinchona powder, and got over the attack in a few days, but remained in broken health for months.¹ His third and last attack (he died in 1847) was at Portsea—also then a malarious place—about the year 1831. This was a very severe attack. He then took quinine, I think in six-grain doses, repeated frequently, as the suffering compelled him. The cure was speedy. He, of course, spoke freely of his success to his brother officers, and, among others, to Dr. —, a naval surgeon of considerable scientific acquirements. Some time afterwards, Dr. —, being in a ship on the Irish station, heard that the Marquis of Anglesea was a martyr to facial neuralgia. He obtained an introduction, and was allowed to make trial of quinine. Probably by a fortunate accident, or perhaps because the drug had temporary power as a nervine tonic, the first effect appeared to be marvellous, and the Lord-Lieutenant knighted Dr. — and settled upon him a very handsome life annuity. The disease, however, seems to have been reflex in this case, being attributed to the condition of the nerves in the stump of the leg which his lordship lost at Waterloo. In that and in other traumatic non-malarious cases of neuralgia, quinine entirely failed.

Neuromata.—In very malarious places in India, as at Chittagong, ganglia, of no extreme sensitiveness, are apt to form temporarily in

¹ I do not know whether it was he who met with the case of a poor woman, living in an English marsh, in whom the pain was so excessive for years that there was a hole in her cheek, attributed to strong pressure with the hand, through which the teeth were visible. Probably there had been *cancrum oris*.

the course of nerves. In one of these cases, a lash of cutaneous nerves, on the inside of the arm of a lady who had suffered much from repeated attacks of intermittent fever, was visibly enlarged with little spindle-shaped ganglia in the course of each nerve, after the manner of a branch of *fucus vesiculosus*. This condition disappeared after some years. In another very remarkable case which my friend, the late Dr. Allan Webb, showed me, there was a much larger ganglion, apparently of this kind, on the pneumogastric trunk on one side of the neck of a Jewess in Calcutta. I have never seen a surgeon who had more complete use of his hands than Dr. Webb had, and he showed me that, by pressing this ganglion—not the common carotid—he could immediately render the woman insensible. The nerve and ganglion were not painful.

ELEPHANTIASIS.

This is a disease which, in its full development, comes within the domain of the surgeon. Its most recently ascertained history has received ample practical exposition from Sir Joseph Fayrer,¹ but elephantoid fever is essentially a 'medical' disease, by early management of which the physician may sometimes hope to prevent the malady from becoming developed into surgical proportions. I have already mentioned that the habitat of Indian elephantoid fever is in marshy places within the range of the sea-breeze, and that it is never developed far up country or beyond the limits of the growth of the coconut tree. Dr. Pringle, cited by Waring, says that, in Cochin, a place so notorious for this disease that the malady is widely known as 'Cochin leg,' there is scarcely a house in which there is not one or more affected; but that, in the interior, at a distance of ten or twelve miles inland, the disease is almost unknown. In one very notorious spot, Waring found 2,133 diseased, in a population of 48,591, or 1 in 21 $\frac{1}{3}$ of the whole population. Fayrer has seen elephantiasis twice only in the pure European in India, Waring neither saw nor heard of a European case. I heard of a European in Bengal, and had under my care a young man, born of European parents in India, but who had never left that country. Dr. Waring believes that brackish water is a cause of elephantiasis. The disease is common in the delta of Bengal to a

¹ 'Elephantiasis of the Leg—of the Scrotum.'—*Clin. and Path. Obs. in India*, pp. 396–409. 'Filaria Sanguinis Hominis.'—*Tropical Diseases*, p. 275. 'Elephantiasis Arabum.'—*Ibid.* p. 258, and *Croonian Lectures*, Lecture Second, 1882. The most valuable of the earlier contributions to this subject are by H. H. Good-ee; *C. M. T.*, vol. vii. part 1, p. 9, and Wise, *Ibid.* p. 156; Allan Webb, *I.* A., vol. ii. p. 619, and an admirable report upon 'Elephantiasis, as it exists in Travancore,' by Dr. Edward John Waring.—*Ibid.* vol. v. p. 1. 'Elephantiasis Arabum, or Cochin Leg,' by Dr. Francis Day. —*Madras Quart. Jour. of Med. Sc.*, vol. i. p. 37.

distance of at least 140 miles above the sea ; but there the sea-breeze prevails. There is a salt lake close to Calcutta. In the freshes, the water of the Hooghly is brackish for some miles above that city, and the water of every well in the delta is also brackish. Webb says that elephantiasis has been seen in the child which could have had no other food than its mother's milk. Waring states that quadrupeds and birds are said to be occasionally attacked in Barbadoes and other places where elephantiasis is very prevalent. In Travancore, he could not hear of any cases in quadrupeds ; but at Allippee, a hen was brought to him in which one leg was very much enlarged ; whether this really was the disease appeared doubtful ; but it certainly bore a strong resemblance to it.

The gravest results of elephantoid fever, enormous integumentary growths, frequently attended with oozing varix of the lymphatics of the parts, vary greatly in different paludal localities. In and around Calcutta, scrotal tumours and elephant-leg abound, and the labia and nymphæ of the female are sometimes attacked. I have seen cases of elephantoid enlargement of the integument of the forearm from the direction of Orissa. The hands are often affected in Travancore. At Chittagong, general as the paludal influence was, I never saw the scrotum attacked, but the prepuce alone was liable to suffer. I removed the growth in two of these cases. In one, the mass, when dissected off, weighed two pounds.¹ This form of disease is occasionally seen near Calcutta. So rare is scrotal tumour in Travancore that Waring found it in only three cases out of 945. He tells me that the Rajah, having become aware of the existence of a case, asked him to examine it as a curiosity. In Bombay, the scrotum is often affected where the prepuce escapes. It appears that, there, the leg is seldom attacked.

When the leg is affected, inflammation, in rare cases, proceeds to sloughing of the whole of the diseased integument, leaving the muscles bare.² In a case, probably of elephantiasis, by Dr. A. Garden, all the structures of the leg mortified.

The history, in all cases, is that the patient suffers from periodical attacks of intermittent, attended with great pain and swelling of the affected part. When the fever subsides, the local swelling is diminished and the pain abates ; but, after each successive attack, the tumour gains some accession of bulk, until, in course of years, the scrotum, for example, may attain a weight of a hundred and fifty pounds and upwards.

¹ Webb considered that, when the disease attacks the prepuce or nymphæ, it is due to syphilis. See case of enormous preputial elephantiasis, 14 inches long and 12 in circumference, in a negro, *æt.* 25, born in Africa, and sent to England from the West Indies.—Wadd. *Diseases of Prepuce and Scrotum*, Plate viii.

² Day.

Waring describes the elephantoid fever of Travancore as characterised by a cold, hot, and sweating stage, constituting together a true paroxysmal fever, except that, generally speaking, the paroxysms are more prolonged and severe in their character, mostly extending over a period of three days, and sometimes longer. There is, in the majority of cases, most distressing tensive headache, lasting throughout the cold and hot stages. The cold is the most marked and severe stage. It lasts one or even two whole days, and all patients speak of it as the most painful and dreadful part of the disease. The hot stage appears also, in most cases, to be very severe. The sweating stage is not always present, but, when it occurs, it is generally by no means mild; the perspiration is described as literally streaming from all parts of the body. Dr. Francis Day had a patient who died from exhaustion occurring at the termination of a paroxysm of elephantoid fever.

Out of 226 cases specially examined, Dr. Waring found a previous history of fever in 224 or 99·11 per cent. In the two cases also, it is probable that malarial influence was at work. Waring shows that, when fever ceases to recur, there is no further enlargement of the hypertrophied part.

All authorities have noticed the fact that, like my attacks of Chittagong fever, elephantoid fever frequently recurs every month. The natives believe that it is influenced by changes of the moon. Both Goodeve and Wise appear to consider that lunar influence is thus operative. Webb calls this fever 'moon fever.'

Writing lately,¹ Dr. S. C. Chatterjee says, 'with every approach of the full and new moon there is generally an exacerbation of fever with intensification of the accompanying symptoms showing increase of the morbid growth.'

Dr. Waring has given a remarkable analysis of 224 cases, in which he was informed that the febrile paroxysm occurred—once monthly in 38; twice monthly in 37; three times in 17; four times in 6; five times in 5; once in two months in 10; once in three months in 24; once in four months in 10; once in five months, 3; once in six months, 9; once in seven months, 2; once in twelve months, 8; once in twenty-four months, 1; irregular in 43; had only one attack, 4; no fever for one year, 2; for two years, 2; for three years, 1; for ten years, 1; for fifteen years, 2. Dr. Waring adds that, however diversified may be the interval between the attacks, yet, in the majority of instances, the febrile paroxysm is stated to recur in the same individual with great regularity. A few observations—say at Cochin—in cases where the attacks are monthly, with a view to ascertain whether they have, in reality, any coincidence with the changes of the moon, would either dispel a popular error or establish a remarkable law of periodicity.²

¹ *I. M. G.*, June 1, 1880.

² A review of the early literature of this important question will be found at

It does not appear that great splenic or hepatic enlargement has been noticed in any case of elephantiasis. But Webb said that, in the fatal cases which he examined, 'disease of blood-making organs, as the spleen, &c., has always been found.' He also observed that 'disordered excretions, particularly that from the kidney, occur during rapid progress of the disease.'

Reports of careful microscopical examinations of the diseased structures have been cited by Webb and Fayrer. 'They show,' to use the words of the latter authority, 'that, though there is a great disturbance in the proportions of the histological elements, there is, after all, nothing really new.' There is, in fact, 'simple hypertrophy of the elements progressing most rapidly and conspicuously in parts, like the scrotum, labia, or certain portions of the integument where lymphatics most abound.'

The existence of filariæ in the blood of these patients, both in the nævoid or lymphatic and simple forms of the disease, has been proved by many observers; but, as these nematodes are not only present in persons apparently in perfect health, but also in association with many other forms of disease, it cannot yet be said, with certainty, that the parasite has any special relation to this malady; still, our well-ascertained facts are bringing us to the verge of such a conclusion. Thus Lewis, Fayrer, Manson, Vandyke Carter, and others, afford us the following data:—chyluria, more properly lymphuria, is a lymphatic disease, so is elephantiasis. In both, there is an exudation of lymph, the loss of which 'exhausts the patient, just as the loss of so much blood would exhaust him.' In lymphuria, the presence of filariæ is constant. Vandyke Carter has seen filariæ in elephantiasis. The increment of this latter disease is periodic. Sonsino and others observe that filarious lymphuria or hæmato-lymphuria is intermittent, with long intervals of apparently complete recovery. It now remains to be shown whether these parasites *always* attend elephantiasis. Fayrer, having clearly set forth these data, judiciously deprecates hasty generalisation upon them. More information regarding the association of filaria and elephantiasis is needful, and may be expected from India, especially upon (1) Manson's remark, that the geographical distribution of elephantiasis is much the same as that of the mosquito; and upon (2) Sonsino's conclusion that, although apparently the intermediate host of filaria, the mosquito probably plays with these para-

p. 113 of Dr. Henry Goodeve's sketch of the 'Progress of European Medicine in the East'—*C. M. T.*, vol. viii. part 2—a memoir of the greatest interest and value to Indian physicians. Dr. Balfour wrote much on the effects of sol-lunar influence in fevers. His first paper appeared in 1789. A very interesting series of 'Cases Illustrative of the Influence of Lunar Agency in Various Chronic Diseases,' was published in 1839, by Mr. B. J. Murray—*I. M. J.*, vol. iv. p. 570. See also p. 24 of the same volume for a very careful review of the question, and a paper 'On Lunar Influence in Malarious Fevers,' by Dr. W. J. Moore—*I. M. G.* for June and July 1869.

sites a part similar to that of birds with respect to vegetable seeds. Again (3), upon the suggestion raised by that authority,¹ do lymphangitis, and adenitis, and lymphatic dilatations and rupture, with extravasation and lymphorrhagia (as in lymphuria and lymph scrotum), depend upon obstruction of lymphatic channels? Also (4), upon the question, Is lymphuria of *frequent* occurrence in elephantiasis?

Few of my readers can have failed to study with great interest Dr. Manson's most able explanation of the highly probable train of events, by which he argues that elephantoid disease is caused by embolism of lymphatic glands by ova prematurely expelled by filariæ.² Dr. Manson shows that the parent filaria is normally viviparous, but that, like other animals, it is liable to miscarry, and the immature ovum which, rolled up, inclosed in its chorion, is of much larger diameter than the long sinuous body of the matured embryo, becomes an embolus. He gives two cases—1, of lymphatic œdema of the legs; and, 2, of lymph scrotum—in which ova of filaria were found in the lymphatics. This is, he believes, the true pathology of elephantoid disease: '1st, parent filaria in a distal lymphatic; 2nd, premature expulsion of ova; 3rd, embolism of lymphatic glands by ova; 4th, stasis of lymph; 5th, regurgitation of lymph, and partial compensation by anastomosis; 6th, renewed or continued premature expulsion of ova; 7th, further embolism of glands.' This process, he adds, 'according to the part of the lymphatic system it occurs in, the frequency of its recurrence and its completeness, explains every variety of elephantoid disease.' Mr. Jonathan Hutchinson has argued³ that, between the permanent solid œdema with hypertrophy of the skin, which finally results from recurring erysipelas and true elephantiasis, the differences are only those of degree. He holds that from such cases of erysipelas to true elephantiasis it is but a few steps, and he avows his conviction, 'that we shall make a definite simplification in our nosology when we class the latter disease as in the main a result of chronic and recurring erysipelas,' which he regards as being 'simply a specialised form of inflammatory action, and not, as it is commonly defined, a specific fever.' It appears to me that the recent practical observations, made in China and India, which I have cited above, place erysipelatous œdematous thickening, and all other hypertrophic conditions of the integument, however closely they may simulate true elephantiasis, in progress and in mere external aspect, quite apart, in pathology, from this latter disease, unless it can be shown, as Mr. Hutchinson does not appear to

¹ Epidemiological Society, May 3, 1882.

² *China Customs Gazette*: Medical Reports for the six months ended March 31, 1882, and *M. T.*, February 17, 1883, p. 89.

³ 'On Certain Diseases allied to Erysipelas,' *M. T.*, January 6, 1883, p. 4.

think of showing, that recurring erysipelas, leading to hypertrophy, is caused by filariæ.

Treatment of Elephantiasis.—When we can get a scrotal or limb case early, and duly combat the fever and the local inflammation, principally by quinine and the application of iodine (iodised oil and iodine paint—Webb), and by rest, especially in the inflammatory stage, as well as by mechanical support and bandaging (Dalton), we can often effect so much good that I used to encourage my students by telling them that, when they were in attendance upon patients in independent circumstances, who would take care of themselves and obey instructions, extensive growth might always be checked, and that such cases might be dealt with hopefully.

When the disease occurs in Lower Bengal, I certainly have faith in the efficacy of quinine *in the earliest stage* of these growths, but my friend, Dr. Francis Day, observed, as the result of great experience of the Cochin disease: ‘Anti-periodics I have found of no utility except in their tonic action. I am so convinced, from experience, that large doses of quinine are only a waste of medicine, that I never employ them.’

Fayrer shows that a complete change of climate, far inland to the native; to a temperate climate to the European, is a distinct indication. He observes that it has occasionally been observed, in the rare cases in which elephantiasis affects Europeans, that, returning to Europe, they have, after a time, lost the disease, and almost, if not entirely, any hypertrophic changes that may have occurred. Webb gives the case of a native of Bengal, in whom the disease remained stationary during twenty years’ service at Agra. On his return to Calcutta, he, in a few months, had repeated attacks of the fever, with swelling and inflammation of the scrotum, and, much against his will, had to go back to the Upper Provinces. Thus his disease treated him as my asthma did me on two occasions.

Dr. Day’s Cochin experience fully confirms this observation. A native left that place, elephantiasis having been ineffectually treated. He went to sea, was absent eight years, and returned perfectly restored to health, all signs of effusion having quite disappeared. Shortly after this, elephantiasis again attacked him. A person went forty miles inland, staying two years; the disease quite left him, and, though he returned to Cochin and resided there for a long time, it did not reappear. On the other hand, a child was sent away, above a hundred miles inland, but the disease did not leave her. Most growths are obstinate in growing children; Dr. Day enjoins a tonic treatment, in addition to the change of residence, for the eradication of the cachexia. When the mischief has gone far, as it too generally has when cases come to us, the tumour having attained a permanent weight of pounds, removal by the knife is alone effectual.

Dr. G. R. Ballingall¹ urges early removal by knife when the disease is progressing rapidly and is attended with recurring febrile attacks, to prevent impairment of the constitution.

All operators—Webb, Fayrer, Day, Ballingall, &c.—agree that, with the ablation of the growth, fever generally ceases. Day gives an exceptional case, which proves the rule—elephantiasis existing in another limb, fever recurred after an operation.

Frequently, in Bengal, empirics declare that they can effect a cure without recourse to the knife. They remove a great deal of fluid, either by bandaging or by the application of the *gool*, but without the slightest benefit. Indeed, whatever condenses the tumour, hardening it by superinduced inflammation, and produces adhesion between the integument and the testes, as the *gool* treatment often does, embarrasses the operator, imperils the testicles, renders the operation tedious, and places the patient in great danger of death by hæmorrhage.

I regret that, although the search at first seemed easy, I have not been able to discover the name of the surgeon who first dared, in these cases, to remove the whole integument of the genitals, trusting that nature would, as she does, cover the exposed parts in a few weeks with new integument, extended from pubes, thighs, and perineum, and supplemented by granulation. It will be recollected that, in operating upon the Chinaman Hoo Loo, at Guy's Hospital, in 1831, Mr. Key lost so much time in endeavouring to save the genitals and to dissect out flaps, which would either have sloughed away or reproduced the growth, that the patient died from hæmorrhage. Calcutta surgeons made flaps then, and for some years afterwards—at least up to 1842. Dr. Ballingall leaves flaps where, as frequently happens in the form of disease prevalent in Bombay, the prepuce is unaffected, and the skin at the sides, drawn down from the abdomen and thighs, appears healthy. This, he finds, facilitates healing, and in no case has inflammation reappeared in the flaps. I have never seen, in a multitude of Bengal cases, one in which healthy skin appeared to be available to form flaps. But, of late, Dr. Kenneth McLeod, of Calcutta, has employed a flap operation, successfully, doubtless in cases where there was more or less healthy skin available.

Mr. F. H. Brett, of Calcutta, was the first surgeon who succeeded, in 1837, in preserving the genitals in removing a tumour as large as Hoo Loo's. We sometimes hear of an unhappy native, who, having been thrown down in a fray, is dragged along by the scrotum until the whole integument is torn away. The surgeon may have seen such a case, and may have noticed how well loss of this integument, by infiltration of urine, is repaired; or the flaps may have sloughed in some of his cases, when he must have observed that Nature did far better

¹ 'Scrotal Tumours in Bombay,' *Bombay Med. and Phys. Trans.*, 1859 p. 1.

without them. Still, when we consider that, in many of these cases, the denuded cords are half a yard long, we cannot but admire the presence of him who first performed this operation. A still higher meed of credit is due to my friend and colleague, Mr. Samuel Bowen Partridge, F.R.C.S., for his admirable adaptation of Esmarch's bandage in the removal of these tumours.

I saw my friend Sir Joseph Fayrer remove a scrotal integument which, with its fluids, weighed rather more than the man did. The man weighed one hundred and eight pounds; the mass, with its fluids, two pounds more. I am not sure how large a tumour has been removed with perfect success. Mr. Partridge reported, in 1880,¹ that the wound was cicatrising after the removal of a mass which weighed 111 pounds, exclusive of serum (of which there were doubtless enormous accumulations in the tuniæ vaginales) and blood.

Dr. James Irving has recorded the particulars of a case² in which the excised mass weighed, without its fluids, 136 pounds. The testicles were not saved. Dr. Irving cites the case of a Jew, at Gibraltar, from whom Mr. Hugh Fraser removed a scrotal tumour, weighing 150 pounds. The penis and testes were preserved, and the man made a perfect recovery. This was probably the largest tumour ever removed with success. But larger scrotal growths have been removed unsuccessfully, or with uncertain result. Dr. Irving reported a second case in which he removed a tumour, weighing 152 pounds without its fluids, (later it became customary to weigh the patient before operating). The man lived seventeen days, and did well until the fifth day. His death was caused by severe attacks of intermittent fever and diarrhœa. Mr. Irving cites Samuel Cooper for a case of uncertain issue, in which a scrotal tumour, weighing 'about a hundred kilogrammes,' or 224 pounds, was removed.

Of 193 cases of scrotal elephantiasis operated on in the Calcutta Medical College Hospital, 18·2 per cent. proved fatal.³ In that institution, Mr. Partridge had 46 operations, with 5 deaths, in twenty-one months ending December 1879. Dr. Kenneth McLeod reports⁴ 129 cases, with 23 deaths.

In his 'Treatise on the Diseases most frequent in the West Indies,' published in 1726, Richard Towne says, of Barbadoes, that 'Amputation of the diseased leg has been performed many times, but has always failed of a cure, for the disease constantly takes possession of the remaining leg.' They did not know quinine then.

Sir Joseph Fayrer has shown⁵ that ligature of the femoral artery has been fairly tried in India, but with no permanent improvement:

¹ *I. M. G.* for January of that year.

² *I. A.*, No. 19, p. 70.

³ Fayrer.

⁴ *Operative Surgery.*

⁵ *Tropical Dysentery and other Diseases*, p. 273.

‘The disease returned to its original dimensions when the patient resumed his usual mode of life.’

The natives of Lower Bengal often suffer, in malarial fever, from a very troublesome congestion and swelling of the Schneiderian mucous membrane, which I have seen. In this *Nakra*, they consider that they obtain relief by thrusting a prickly grass up the nostril, so as to cause bleeding—

Bardolph. Yea, and to tickle our noses with spear-grass to make them bleed.—*King Henry IV.*, Part 1, Act. ii. Scene 4.

Dr. Wise says, in his ‘Hindu System of Medicine’ :—‘*A’haka-jwara* (*Nakra* or *Násá-jwara*).—The natives of Bengal are subject to this peculiar form of intermittent fever. The description of it is not contained in the more ancient medical works. It is produced by a swelling of the Schneiderian membrane, and is said to be caused by the air and phlegm deranging the blood of the part. It is accompanied with pain over the body, but particularly in the neck and back, and other symptoms of fever. It is of frequent occurrence in Bengal, attacking adults alone, between the ages of twenty and forty. It is treated by the natives by abstracting a little blood from the nose by the point of a needle, or by rubbing a little rough grass over the diseased part. Errhines are also of use, and those who take snuff daily are free from the disease.’

MALARIAL INFLUENCE IN CASES OF WOUNDS, &c.

It was observed many years since, by the late Mr. Stafford, of London, that, in England, surgical fever is apt to assume the periodic character in patients from marshy districts. We have abundant evidence of this fact in Bengal. Again, Sir Joseph Fayrer gives, in his second Croonian Lecture, some most interesting observations by Mr. Eccles, surgeon of the Stafford House Ambulances during the Russo-Turkish campaign, on the effects of malarial fever on wounds. Sir Joseph’s own experience confirms that of Mr. Eccles. I saw, in my colleague’s wards, the cases of death from *urethral fever* and *gangrene from wasp sting* to which he refers in the following quotation: ‘The mere passing of a catheter in a comparatively healthy man has been followed by paroxysms of fever and deposits of fibrine in the pulmonary artery, which have proved rapidly fatal. The slightest wounds have, in those more deeply affected’ (by malarial cachexia), ‘been followed either by gangrene or by hæmorrhage—the abstraction of a tooth, by necrosis of the jaw; a pimple on the face, by gangrene of the cheek; or the sting of a wasp, by gangrene of a limb—all proving the low vitality and tendency to disintegration of the tissues in this diseased condition.’

A few remarks upon *Urethral Fever*, especially as depending upon paludal influence, as it occurs in India and in England, will be found in the chapter on Diseases of the Urinary Organs.

PUERPERAL FEVER.

It was remarked by Lind, in the last century, when Calcutta and the other presidency towns were very unhealthy, that, in India, European women in general enjoyed a much better state of health than the men, but that child-bearing was, in his time, peculiarly fatal to them in Calcutta; on which account he considered that it would be advisable for them to retire to a more healthy situation at the approach of their delivery. In the absence of more explicit information regarding the prevailing causes of death, we may state the probability that *post-partum hæmorrhage* destroyed many who were the subjects of neglected malarial cachexia. This dangerous accident is still always so much to be dreaded in India that I never omitted to have a sheet-binder placed under the patient from the first, and to give a full dose of ergot as soon as I knew that the child was descending quickly. Clarke, writing in 1770, mentions putrid puerperal fever as extremely common in Bengal. The Hindoo women, who, at the time of their confinement, are, in many cases, placed apart in small, damp, ill-ventilated rooms, artificially heated, are still very liable to puerperal metritis, pelvic cellulitis, and peritonitis. History contains only too abundant evidence of the fact that, in Mediæval England, the lives of even royal ladies were frequently sacrificed under a similar practice of 'lying-in' in unventilated castle chambers, over-heated and scrupulously closed against every indraught of pure air. This was, probably, one of our many customs learnt in the Crusades. In 1836, the surgeon to the Calcutta General Hospital wrote:¹ 'Puerperal fever, in its severe form, is a rare disease in Bengal.' Mackinnon, who, previously to the appearance of his great work, was stationed at Tirhoot, Dinapore, and Cawnpore, spoke of true puerperal fever as rare—he believed totally absent—in India.² Until of late years, within my knowledge, Lower Bengal was nearly free from erysipelas, and puerperal fever was not common in the obstetric wards of our hospital. When erysipelas became very prevalent in Calcutta, in June 1874, I inquired of Dr. Charles whether there was any puerperal fever in his wards. He replied that they were particularly free from this disease. Shortly afterwards, however, so many cases occurred among his patients, that I had, at his request, to provide accommodation for them in a separate building.

Dr. De Renzy states³ that Abbotabad, in the Punjaub, has long

¹ *I. M. J.*, vol. iii. p. 117.

² *Op. cit.* p. 381.

³ *Transactions of the Epidemiological Society*, 1882-83, p. 35.

had a bad reputation for puerperal fever. He thinks this may be attributed to the practice of washing the Europeans' clothing in the watercourse which is the natural drain of the native town.

MALARIAL POST-PARTUM FEVER.

At Chittagong, upwards of thirty years ago, and also in Calcutta subsequently, I had in my practice frequent illustrations of the fact that, there, soon after delivery, both European and native women often get fever, generally with arrest of the milk and lochia. At first, these symptoms were equally terrifying to the patient and the physician; but I soon found that no other severe symptoms were developed, and that quinine speedily restored health. I had many of these cases; but not one that did badly or long refused to be controlled by the specific.

I was much interested, in 1880, by reading¹ the report of a discussion upon this fever as it occurs in the neighbourhood of New York. It is called by Professor Fordyce Barker 'puerperal malarious fever.' As in India, it stands quite apart from all the forms of deadly 'puerperal fever.' I prefer my own nomenclature for the Bengal malady. Professor Barker observes that it may occur from twenty-four hours after a normal labour up to completion of puerperal recovery. Only one of his seventeen cases was fatal, on the forty-seventh day after confinement. He gives half-ounce doses of Warburg's tincture every four hours until the fever is entirely abated, continuing it, in diminishing doses, until convalescence is perfectly established. He finds this a far more effectual and speedy remedy than the largest doses of quinine. In the discussion which followed the reading of Professor Barker's paper, Dr. Jones, who lives in a malarious district, said that he gives a dose of quinine on the day following confinement. Dr. Lusk considered that it is needful to remove these women from lying-in hospitals, as puerperal peritonitis and cellulitis begin to appear in other cases. Dr. Barker puts his patients on quinine immediately after confinement, from January to June, and continues it for ten days. Dr. Emmett referred to cases in which there were uterine hæmorrhage, sub-involution, cellulitis, &c., in women who had been living in malarial districts, and which could not be cured except by quinine. Professor Barker does not mention the lochia. Dr. Gillette speaks of 'the purity of the lochial discharges' as a diagnostic point in these cases. In the Bengal disease, as I saw it, the lochial discharge was checked until the fever abated.

In 1839, Dr. Duncan Stewart, then of Calcutta, published² some very important remarks upon the predisposition of pregnant women

¹ *M. T.*, March 20, 1880, p. 327.

² *I. M. J.*, vol. iv. p. 587.

to fever, and upon the great danger of *Bengal fever when occurring late in pregnancy*. He shows that it generally happens that, when labour occurs in the course of fever, it is *premature*. He cites the following striking observation from Twining: 'I hardly deem any part of the observations I have to offer concerning the peculiar nature and treatment of the remittent fever of the rainy season in Bengal of more importance than the caution I have now given respecting the hazard of either bleeding or applying leeches at a late stage of the paroxysm, after the disease has been returning daily above a week, and when the patient is much exhausted. I have met with several instances where the application of leeches was, from trivial causes, delayed for three or four hours after they were ordered, and then put on towards the conclusion of the paroxysm when arterial action was subsiding—the patient being in a state of languor and anxiety, and the skin perspiring freely—and death has been the consequence.'

'If,' says Dr. Stewart, 'such results have followed the loss of a minute quantity of blood under ordinary circumstances, will it surprise anyone that the occurrence of even natural labour at the end of a paroxysm of fever should be matter of more than ordinary anxiety, or that the abstraction of so large a portion of blood as must attend even the best-conducted delivery should, at such a time, be eminently perilous?'

I have seen grave remittent fever, in Bengal, passed through under quinine, by which a cure was effected some weeks before delivery, with perfect immunity both to mother and child; but a more anxious case for the physician it is hardly possible to conceive.

I cannot find a more appropriate conclusion, not only for this particular subject, but also for my review of Indian malarial fevers, than the following striking passage from the writings of Dr. Barnes:¹ 'The pertinacity of ague is well known. We are accustomed to see marks of its abiding influence long after the fits have ceased, long after the patient has lost all consciousness of suffering. But I have seen ague fits reproduced years after under the condition of pregnancy. We cannot fail, then, here to see that there lingers somewhere in the system some change, the stamp of the original disease; and we may fairly presume that this change is in the nervous centres. Perhaps the precise organic change may long baffle the skill of the microscopical analyst to detect; but there it is proved by anticipation to exist, as real a thing as the planet discovered by Adams and Leverrier before it was seen by mortal eye through the telescope.'

¹ 'Inaugural Address,' *M. T.*, August 18, 1877, p. 168.

QUININE IN THE FEVERS AND OTHER DISEASES OF INDIA.

Close observation will always show that, in every Indian fever, hectic, the true enteric fever of Jenner, measles, small-pox, erysipelas, puerperal and surgical fever, the malarious taint is present and must be dealt with. Whatever disease one who has resided in the plains of India may be the subject of—quite irrespective of the time, however long, which he may have subsequently passed in a temperate climate (which is, probably, only less malarial than India)—the necessity of giving him quinine must not be forgotten. In uniformly pursuing this course, no disappointment has ever daunted or discouraged me, my conviction being that I have worked in the right direction. True, quinine has not the power of cutting short an attack of true enteric fever or of curing the hectic which attends phthisis or hepatic abscess; but no medicine at our command has so valid an influence as it has in maintaining vitality and in moderating the temperature in these diseases as they occur in India. I have noticed above the fact that quinine puts down the fever which not unfrequently appears after child-birth in India and other malarious countries. In cases of true puerperal fever, quinine is also indicated, but will not display equal curative energy. In England, the value of quinine in erysipelas—here, as in India, the congener of puerperal fever—has long been recognised. In like manner, the fever which follows a surgical operation in India and elsewhere is, as was long ago shown by Mr. Stafford, of London, sometimes periodic and curable by quinine. Stafford has noticed, in England, that urethral fever has occasionally assumed the character of an intermittent, the patient doubtless being the subject of a malarious taint. If the surgical fever be that of septicæmia or erysipelas, quinine is indicated, but may not be equally valid. We are all in the habit of treating every disease with especial regard to any constitutional taint which may affect the patient. Nearly everyone who has lived in India must carry with him until he dies the taint of the paludal poison. Consequently, treating him for any disease in the nosology, we shall treat him inefficiently if we overlook this fact. In *any* difficult case in India, I have always stood prepared to give quinine, never in heroic doses, but in frequent doses of from three to six grains. Thus employed, I have never seen it do the slightest harm in any case, and have never been compelled to discontinue its use until the desired effect had been produced, or to regret that I had invoked its aid; and I have no doubt that those who have practised in malarious districts at home have generally made the same observation.

Note on the Subcutaneous Injection of Quinine in Malarial Fevers.—My friend, Brigade-Surgeon J. B. Scriven, read a highly

valuable summary of his large experience in this practice at the International Medical Congress at Amsterdam in September 1883. He shows that the use of a solution of thirty grains of quinine in half an ounce of water, with eight or ten drops of dilute sulphuric acid, was potent in checking the fever, but led to numerous ill-results, such as abscess, ulceration, and tetanus. Consequently, this mode of using quinine was prohibited in the European army in India. Mr. Scriven also abandoned its use in natives for the same reason. Subsequently, Dr. J. E. T. Aitchison and Mr. Scriven had remarkable success in the use of a solution containing quinine a drachm, tartaric acid half a drachm, and distilled water sufficient to make three fluid drachms, Buzzard's syringe being employed. He was careful to select a spot, usually at the back of the forearm, where no cutaneous vein became visible after grasping the arm above the elbow, and to introduce the point of the instrument with the opening *away* from the skin, so that the solution should escape towards the muscles instead of towards the surface. He employed this method for four years, in about eighty cases, without a single instance of tetanus, abscess, ulceration, sloughing, or any untoward result. As a rule, a little inflammation of the forearm followed. The arm being kept in a sling and cold lotions being applied, this subsided in a few days. In an adult, Mr. Scriven injects from fifteen to twenty minims of the solution—fifteen minims containing five grains of sulphate of quinine. In one case, of a child under a year, he injected seven minims. He especially recommends this practice in cases in which death is imminent, and a remedy is necessary that shall produce a rapid and decided effect. Mr. Scriven informs me that he has adopted this course 'in several cases of fever in which there was great exhaustion, the surface covered with a cold perspiration, the pulse scarcely perceptible, and every appearance of approaching death, and these cases have recovered without any recurrence of the fever.'

CHOLERA ASIATICA MALIGNA.

The cholera of Lower Bengal, of which I had more than twenty-seven years' experience, has been so fully and faithfully represented in works of great labour and research by several contemporaries of my own, that I shall not offer a description of its general characters which will be found amply detailed in the valuable monographs of John Macpherson ('Cholera in its Home,' and 'Annals of Cholera'), of Edward Goodeve (art. 'Cholera' in Reynolds's 'System of Medicine'), and Charles Macnamara. I shall, therefore, only submit to my readers notes upon very important points which have received my special attention.

Cholera a Pernicious Fever.—A great body of well-ascertained facts supports the position that cholera is a pernicious fever. Many Indian observers have maintained this view, and careful study of disease in India goes far to confirm it. I have here placed before myself the duty of stating facts, not of forming theories, or of discussing the theories of others, except by showing how far well-ascertained facts uphold or are opposed to them. I do not consider that the present state of our knowledge permits others or myself to write dogmatically upon the perplexed question of the cause of cholera, but I am strongly led, by observation and reading, to side with the maintainers of the fever doctrine.

In previous chapters, I have described several forms of pernicious fever, which certainly have the appearance of forming a continuous chain of clear, intermediate etiological links between the malarial remittent of India and Asiatic cholera. I will beg the reader to place marks at pp. 169, 181, 184, 189, 190, 191 and 249, and to look back to these cases and observations as I shall now refer to them.

1. To me it appears that Allan Webb's cases of so-called 'Hill Colic' (p. 189), and my case of the Mohammedan (p. 190), may be taken as forming the first links in this connective chain. Here we have the strongest expression of algide pernicious fever, with intestinal lesion, but without cholera stools; rapid death by nervous shock and blood-poisoning, with collapse as its main characteristic—vitality failing almost suddenly under an overwhelming dose of a strong poison.

2. Next in order, we have cases of well-marked and fully developed remittent fever, by no means rare in India, represented by Dr. Sutherland's case (p. 169), and by Mr. Raleigh's cases (p. 181), taking on the diaphoretic algide form—collapse threatening death, with sweating, but without vomiting, and not always with purging, at the end of a regular paroxysm of fever.

3. The next link is formed by certain so-called cases of 'hæmorrhagic dysentery' (which, however, stand quite apart from true dysentery), not infrequent in India, where paludal fever displays the pernicious character by an enormous flow of disintegrated blood from the bowels with collapse (*vide* Mr. Hare's case, p. 190).

4. Then come cases, undoubtedly of fever, with heat of skin, with or without vomiting, and with copious discharges of thin sanguinolent fluid, such as the following, by Baboo Odoy Chand Dutt :¹—A native prisoner, aged twenty, was admitted with intermittent fever, not apparently very severe, of six days' standing. The next day, it was reported that he had fever throughout the preceding day and night, which remitted. On the third day, there was the same report. At 4 P.M. he had three copious watery stools of a bright red colour, very thin, without large coagula. He became pulseless and collapsed, and

¹ *I. M. G.*, February 1869.

died in an hour. The peritoneum and large intestines generally were congested. About a pint of reddish serum was effused into the abdominal cavity. Cæcum congested, thickened, and covered with red patches. Large intestines filled with a dark-yellow fluid.

Six months later, a prisoner, aged thirty, complained of fever for two days, for which he only sought admission to hospital on the third day. He had vomiting and purging that morning. In the afternoon, he had two copious watery stools of a deep dark-red colour, with flocculent yellowish deposits, and vomited thrice—bile and mucus. He was found with sunken eyes and very feeble pulse, abdomen sunken and free from tenderness, very restless. He had one scanty stool at night of a deep red colour. On the following morning, collapse had passed away, but there was sickness and retching. He had strong fever at noon, which left him in the evening with a weak pulse. On the next day, he had fever with sickness from noon till 4 P.M., less strong than on the preceding day. Quinine had begun to tell. On the following day, he felt pretty well, but very weak. He had no further bad symptoms. See Mr. Foster's case of an artillery-man, p. 249.

5. I twice saw, in Bengal, outbreaks of a disease which wanted none of the usual characteristic symptoms of true Asiatic cholera, except that the rice-water stools were blood-tinged. [In another similar outbreak I would recommend careful thermometrical observations.]

It is shown, in the annual report of the J. J. Hospital, Bombay, for 1862,¹ that, in seventeen cases of cholera, all of which were fatal, the stools were of a pink colour, apparently from 'partial transudation of the colouring matter of the blood.' In several of these cases, the mucous membrane of the small intestines 'was unusually red, and the solitary glands in a marked manner prominent.' In the first appearance in my ward of cases of this type, all did well. When, after an interval of years, some more cases appeared, I was very hopeful, but all died. In the museum at Haslar Hospital, there used to be a dried preparation of the lower part of the ileum taken from a patient who died in the first epidemic outbreak of Asiatic cholera in England in 1832. For about a foot above the cæcum, it was of a deep mulberry-red colour.

In the only outbreak of truly *epidemic* cholera that I chanced to see in Lower Bengal, in 1849, I found, in all of my numerous fatal cases, intense redness of the lower part of the small intestine (over which there was great tenderness during life). The stools were rice-water-like without the slightest tinge of blood. This was accounted for by the presence of a condition which I have never again met with in cholera. The congested mucous membrane was evenly coated by a strongly adherent, croupous-like, white exudation, nearly as thick as the intestine itself. From the inner surface of this, thin white processes floated convergently towards the axis of the canal into shreddy rice-water

¹ *Bombay Med. and Phys. Trans.*, No. 8.

fluid. Clearly, all passage through the inflamed gut had ceased some time before death. Mentioning this to the intelligent Madras dresser who assisted me in my military hospital, I was told that the regiment had suffered from such a type of cholera in Madras several years previously ; and he showed me, in some of the older men, huge cicatrices in the right iliac fossa, evidently caused by a powerful escharotic. I counter-irritated so decidedly in the case of our chaplain that, for years afterwards, he struck his side whenever we met.¹

6. At p. 19, I have given cases by Dr. Geddes and myself, which might be regarded as examples of true cholera, save that rice-water stools occurred in the course of fever.

Under this head, we must class a type of fever (some considered it to be true cholera—which it assuredly was not) described by Dr. Murray, in 1840,² as the ‘Malwa Sweating Sickness.’ The attack commenced with rigors or chilliness, followed by dull headache, increased heat of skin, and dilated pupils, a burning sensation at the epigastrium, with restlessness and thirst, and generally copious watery motions smelling like the flesh of carnivorous animals slightly tainted. In many cases, there was vomiting of a similar fluid, with cramps in the extremities ; and the skin soon became bathed in perspiration. There was great oppression in breathing, with anxiety and præcordial uneasiness and weak rapid pulse. At the commencement, there was prostration of strength, with a feeling of exhaustion ; and, afterwards, there was real debility, sometimes extending long into convalescence. In the severest forms of the disease, all bodily uneasiness soon ceased, except that arising from the thirst and the pectoral oppression ; the perspiration continued excessive and became cold. The mental faculties remained clear until towards the end, when, coma gradually intervening, death sometimes ensued within ten hours of the commencement of the attack. Vomiting and cramps were neither constant nor prominent symptoms ; but, in the severe cases, no urine was passed, nor was there any bile in the evacuations till reaction ensued.

When the disease took a more favourable turn, the pulse became more full and the præcordial oppression diminished ; some dark green fæculent matter was passed by stool, a little urine was secreted, and the patient slept. If the case did not proceed at once to convalescence,

¹ Although he does not describe this type of cholera, Annesley refers to this mode of treatment (*Sketches*, 1829, p. 157): ‘A nitric acid blister has been strongly recommended, and I have given it a full trial,’ . . . ‘it always made an eschar.’ ‘Scalding water has been recommended as a blister.’ As, in cholera, the gravest incidence of disease is always upon the lower part of the ileum, I think that we should never fail to apply strong counter-irritation over the right iliac fossa. I always did so.

² *Madras Quar. Med. Jour.*, vol. ii. p. 77, and vol. iii. p. 807.

the pulse did not become natural, the pupils remained sluggish, there was anxiety, and the skin continued muddy and strongly perspiring.

After a remission of twenty-four or forty-eight hours, sometimes anticipating by two hours, the same train of symptoms was apt to be renewed. The skin became dry at first, and sometimes hot; the burning sensation in the epigastrium recurred, followed by two or three watery nauseous stools, and great exhaustion of strength; and, although the skin became cold, the perspiration increased. There was occasionally wandering of the mind, but extreme collapse, with a state approaching coma, was more common; and these increased after each periodic exacerbation or paroxysm if the case was proceeding unfavourably. There were never any cramps after the first attack, and vomiting was also less frequent. The appearance of the stools was brown, green, or yellowish—the latter being most favourable.

As the disease went on, remission succeeded the paroxysms with a regular periodicity. When the patient was to recover, the attacks became more slight, and sometimes convalescence was rapid, without leaving any organic derangement; but, when the disease was of a dangerous character, the collapsed and comatose states were more prolonged after each exacerbation, and sometimes the patient never rallied after they came on. One patient remained three days in a state of coma, yet ultimately recovered; in one case, convulsion preceded coma, and, in two others, it came on after very acute pain in the region of the kidneys, which appeared to be spasmodic, as it subsided suddenly. In several cases, uneasiness was complained of about the heart, and continued for some time after convalescence was established. Dr. Murray was strongly inclined to think that this was caused by the formation of coagula in some of the heart's cavities.

In commenting on the above report, Dr. Corbyn wrote, very curtly, that this disease, 'of a periodic remittent character, attended with a peculiar foetid perspiration connected with a depraved condition of the blood, is nothing more than a modified form of cholera which has occurred in various situations in India, but especially at the Presidency (Calcutta). This disease (which Dr. Murray, senior, designated 'febris remittens choleroidea') has, however, shown itself repeatedly elsewhere, and has been generally recognised by those who witnessed its ravages as a pernicious type of remittent fever.

In the following example, this fever appeared during the prevalence of cholera in the regiment, and may almost be regarded as a hybrid of cholera. This history is one of singular interest, as showing the close relationship which exists between pernicious remittent and cholera.

In November 1840 (nearly contemporaneously with the appearance of the 'Sweating Sickness' at Malwa), a wing of H.M.'s 4th Regiment were in camp, cholera having prevailed at headquarters, near Bellary,

an inland station centrally placed between Madras and Bombay. Drunkenness was prevalent, and the men were exposed to the sun and to the chilly nights without adequate clothing. Throughout the preceding month, there had prevailed an aggravated form of remittent fever 'of a highly sthenic character, presenting many of the features of the ardent remittent of the West Indies.' The fever now, however, became congestive, manifesting a strong tendency to terminate either in sudden cerebral effusion or in prostration and collapse. Mr. W. Parry said that, in most instances, it was ushered in by dull pain in the head, giddiness, nausea, oppression at the præcordia, thirst, restlessness, and a feeling of extreme exhaustion; the countenance was either dull and heavy, or anxious. There was seldom much febrile reaction, and the pulse was mostly very weak and small. Death was ushered in by sudden prostration and collapse, sudden sharpening of the features, and a severe, but indefinable, sense of uneasiness and suffering at the præcordia, attended by frequent sighing and extreme jactitation. Then two or three watery evacuations were passed in rapid succession, and these were instantly followed by extreme collapse. The body became, all at once, deadly cold and covered with a clammy, unhealthy sweat. Pulse almost imperceptible. Drowsiness supervened, and soon terminated in fatal coma. The above was the type assumed by the disease in worn-out and intemperate men. In more youthful and unbroken constitutions, and indeed in the more ordinary type of this fever, there was generally much greater febrile and vascular development, and a greater tendency to assume a very irregular periodic type, most frequently an irregular quotidian or double tertian. In two men, after what appeared to be a complete intermission for two days, the paroxysm commenced with severe convulsion—in one, ending in paralysis and coma, fatal the next day; in the other, merging in long-continued stupor, which passed off by very slow degrees. In other cases, in which the disease had observed considerable regularity for three or four days, instead of a recurrence of the regular paroxysm at the anticipated hour, there was sudden collapse, the body became cold and covered with clammy, unhealthy sweat, the limbs and countenance purple, and the circulation nearly arrested. This profound collapse occurred in one individual at nearly the same hour for three successive days, after which he gradually recovered.¹

At page 184, I have cited the description of the leading features of an outbreak of pernicious remittent, which occurred in H.M.'s 6th Foot, stationed at Deesa, Bombay Presidency, in September and October 1835. In many of these cases, there was deadly collapse.

'With this sudden sinking there sometimes existed internal heat and thirst. The extremities were often deadly cold, while the trunk was warm.' In some cases there were 'evening exacerbations, somno-

¹ *I. M. J.* for 1842, p. 389.

lency, immobility of pupils, or bilious vomiting and watery purging two or three days before death.' 'In other cases, of considerable severity at first, there were nocturnal perspirations so profuse as to drench the bedding.' Dr. R. Brown also described this outbreak as it occurred, at the same time, at Deesa in his troop of Horse Artillery. His description of the disease fully agrees with that by Dr. Jackson, of the 6th Foot, which I have cited. After two or three days of suffering from a sense of extreme exhaustion, there would be a few rigors, with shrunken features, oppressive headache, epigastric weight and tenderness, with much nausea and ineffectual retching, followed by some reaction, raised temperature, acute headache with occasional delirium, bowels generally torpid, nausea constant, retching distressing, sometimes followed by vomiting of dark-coloured fluid. This stage was very uncertain in duration, sometimes partially remitting and again returning. At length, perspiration would break out, happily sometimes warm and general, bringing relief of all the symptoms for a time: sometimes, not unfrequently, partial, cold and clammy, without relief of symptoms, with continued irritability of stomach; feeble, quick, compressible pulse; great depression of spirits; occasionally involuntary passing of watery stools; urine scanty, dysuria. In many cases, this condition, which seemed almost to amount to irretrievable collapse, continued for a long time, and would be succeeded by a reaction, generally less strong than the previous one, but marked with symptoms of congestion of liver, spleen, or brain; and this process would be again gone through until the disease was subdued.

7. Dr. Bellew has given¹ an account of the 'cholera fever' which prevailed in 1880 at Amritsur. 204,385 patients were treated between October and December in six hospitals. The mortality—August–December—was 14,480—nearly three times the mortality of the preceding year. There were three types: (1) a malarial fever, prevailing mostly outside the city; (2) a continued fever, of virulent type, which raged within the city; and (3) cholera. In many cases, the last two types were mixed or combined, so that the same patient presented symptoms of both the prevailing fever and the prevailing cholera. The malarial type yielded readily to the usual treatment. The virulent continued type was limited to the city. It presented many of the symptoms of relapsing fever. These were rigors, fearfully severe headaches, insomnia, disordered bowels, pyrexia, suppression of urine; and death by coma often supervened with terrible speed. Often, within a few hours of the advent of the fever, symptoms of cholera—vomiting and rice-water stools—appeared, and 'it was simply impossible to pronounce, in these cases, whether the disease was fever or cholera.' The sequelæ, in those who recovered,

¹ In his 'Report of the Sanitary Administration of the Punjaub for 1880,' cited in the *I. M. G.* for July 1882.

were enlarged spleen, anæmia, debility, jaundice. The fever was considered to be infectious; whole households were prostrated at the same time, and those whose duty took them within the city contracted it. Quinine and other antiperiodics had no influence over it. Apparently, the causes of this terrible outbreak were, an unusually heavy monsoon rain, bad city drainage, and an evilly worked system of dry conservancy.

8. Last in this progressive series, which has not, I believe, been thus fully traced out by any preceding observer, is the fully developed expression of pernicious fever in malignant Asiatic cholera.

Several authorities—Hutchinson, Searle, and others—held, long before I went to India, that cholera is fever, the gravest development of the potency of marsh poison. Many of our data are in favour of this opinion, and I doubt if there is any fact in the history of cholera which is irreconcilable with this view. At the same time, I do not think that, considering the large means of research, upon the lines so ably followed by Drs. Lewis and Cunningham, still open to us, we should be justified, at present, in adopting this or any other theory of the cause of cholera. I shall continue to watch the quest for the 'cholera germ' with a perfectly open mind, equally prepared to learn that, if such an organism really exists—which appears most doubtful—it is a hitherto undetected microzyme, or the at present rather slighted bacillus *malariae*:—not, until after its claim to recognition as a causative agent shall have been further sifted, the comma bacillus of Koch.

If we accept the opinion that cholera is a pernicious fever, we must not expect to find that it is either contagious, as small-pox is, or that it is propagated from individual to individual, as a specific poison, as syphilis is.

All my large personal experience of the disease in Bengal—its home—and all that I have read, confirm my belief that the essentials to its occurrence are (1) an atmospheric or telluric condition, epidemic or endemic, due, it may be, to some undetected abnormality in the air or in the earth—electric, volcanic, or other—or to the presence of some undemonstrated microzyme or germ, which predisposes those who live within the area of its occupation to cholera; and (2) an exciting cause such as panic, fatigue, want, or unwholesome ingesta—say, a dose of Epsom salts, putrid fish or meat, crude fruit, or water impregnated with various impurities, such as cholera excreta, &c., &c., &c. No one has proved, to my satisfaction, that cholera stools contain a *specific poison* capable of propagating cholera to those who swallow it. True, some of the facts which are adduced in support of their doctrine by those who hold that cholera is propagated exclusively by cholera stools are striking; but these facts are very few, considering the great prevalence of the disease and the vast number of those who have studied it practically; and, carefully sifted, the strongest of them fall short of

absolute demonstration. Thus great weight is given to the fact that in one of the later visitations of this metropolis by cholera, the ravages of the pestilence were signally incident upon the East of London. Upon special inquiry, more than one of our best sanitary authorities discovered that, when the disease was most active, the inhabitants were drinking unfiltered water from the river Lea; and that, at the same time, this water had received cholera stools. This is a showy fact, but it quite falls short of demonstrating that a specific poison in water caused this outbreak, seeing that, whenever pestilence has visited London, its incidence has always been most heavy at the East End, where poverty and sanitary neglect, in a marshy atmosphere, have constantly prevailed,¹ and that filthy drinking-water has always been recognised as a valid *exciting* cause of cholera—as, I repeat, a strong solution of Epsom salts also undoubtedly is.

Mr. Macnamara states,² that the following remarkable accident occurred within his own observation:—A small quantity of a fresh rice-water stool, passed by a patient suffering from cholera, was accidentally mixed with some four or five gallons of impure water, and the mixture exposed to the rays of the tropical sun for twelve hours. Early the following morning, nineteen people each swallowed about an ounce of this contaminated water. Within thirty-six hours, five of these persons were seized with cholera.

This again is a striking fact. Still, it quite fails to prove that the rice-water cholera stool contains, and is capable of communicating, when swallowed, the specific poison of cholera, as the virus of syphilis and variola contain, and, when inoculated, communicate, those two diseases. Indeed, the fact that scarcely more than 25 per cent. of those who swallowed the poison became the subjects of cholera may be received with surprise by some that so many escaped, may be viewed by others as an evidence that no poison was swallowed, and must be received by all as proof that, if cholera stools do contain the specific poison of cholera, that poison is far less sure in its action on the system than various recognised specific poisons are. Thus, if three

¹ In illustration of this point, I may as well quote, *verbatim*, the words which I used, in November 1882, (*M. T.* for November 11, 1882, p. 577), when speaking of the general unhealthiness of East London, without having my attention at all particularly directed to the subject of cholera:—‘We read that, in the first year of Charles the First, Stepney lost 2,978 persons by the plague, and that, in 1665, plague again appeared there, and, according to the parish clerk’s account, swept off 6,583 persons.’ . . . ‘It is stated, in the “Life of Lord Clarendon,” that “the plague had swept away so many seamen (Stepney and the places adjacent, which were their common habitation, being almost depopulated) that there seemed to be an impossibility to procure sailors to set out the fleet.” Stepney lost a character for salubrity, which it had somehow obtained, when, in 1844 and 1866, many cases of cholera occurred in its neighbourhood.’

² ‘Cholera,’ Quain’s Dictionary, p. 240.

groups of persons (1) took scruple doses of arsenic, were (2) inoculated with small-pox, or (3) with syphilis, it can hardly be doubted that more than 25 per cent. of each party would manifest the specific action of each of these three poisons.

Then, should it be argued, by one unacquainted with the natural history of Indian cholera, that the fact that, in a cholera outbreak, so many as five out of a party of nineteen were attacked with cholera creates strong suspicion of direct cholera poisoning, I shall presently give a case, by Dr. Macpherson, in which five out of eighteen inhabitants of a house were attacked with cholera, three fatally, when there did not exist any evidence or suspicion of direct cholera poisoning. So, too, many cases have been adduced in which pilgrims, travelling in the course of a cholera epidemic, reached a town and were attacked with cholera, whereupon cholera appeared in that town. To this argument of the propagationists their opponents reply by inquiring, Would not the epidemic have appeared in the town, precisely as it did, if the pilgrims had never come? Does the admission of a cholera patient to a hospital impeach that hospital with cholera?

In considering this question, it is noteworthy that nearly the whole of those Indian authorities who believe that cholera is communicable by the dejecta judge from their experience of *epidemic* cholera in the North-Western Provinces; whereas nearly all who have long worked in Lower Bengal, the fixed *endemic* area of cholera, doubt that this disease is at all communicable from man to man.

Thus, only a day or two before this chapter was written, a brother officer, of remarkable distinction, who has retired after long service both up country and in Lower Bengal, told me that he long sought in vain for an instance of a servant who had got cholera in the wards of a cholera hospital. At length, he found a *napit* (barber) who had been attacked under what appeared to be suspicious circumstances. To the inquiry, 'Have you been shaving cholera patients?' he replied, 'I never shaved a cholera patient.'

During fifteen years, with some breaks, in which I was senior physician of the largest hospital in India which was rarely free from cholera cases, no medical officer, nurse, or native hospital attendant (altogether a large body) was attacked with cholera. When I first assumed charge, *all the cholera cases were treated in the general wards*, side by side with the other sick. After some years, I fitted up a cholera ward for men; but, up to the day on which I left India, I could never obtain separate accommodation for the women. Still, in those fifteen years of never-ceasing cholera prevalence, I can recollect no patient who was attacked with cholera in hospital, with the exception of Willie Marshall, a little patient of my own, and a native man who recovered. Therefore there is nothing, in my own experience,

that favours the opinion, entertained by many, that every cholera patient is to be regarded as a centre from whom pestilence radiates.

At a meeting of the Epidemiological Society, in the summer of 1883, Dr. Cuninghame stated that he had collected nearly eight thousand cases of cholera attendants, of whom only 150 were attacked—these, like all their neighbours, having to share the danger. Dr. De Renzy replied that it had been known that soldiers, told off to attend their sick comrades in the cholera wards of European corps, suffered largely. Here it should be borne in mind (1) that, when epidemic cholera attacks a European regiment, *all* are in great danger; that, (2) gallant as the English soldier generally is in battle, and devoted as he is in attention to his sick comrades, the first outbreak of cholera pestilence always raises panic—a strongly predisposing cause—throughout the regiment, especially when the medical officers are propagationists; whereas (3) work with cholera patients, which he knows by experience to be unattended with the slightest danger to himself, is the daily business of the native hospital attendant, who, although not courageous, generally has fair good sense.

At the Calcutta Medical College, I had a little town within my walls, numbering, at mid-day, upwards of two thousand inhabitants, a very large majority of whom were in some way concerned with our very numerous cholera sick. Our sanitary arrangements were fairly good, but very far from perfect, seeing that the hospital was a remarkably faulty building, standing in a nice, but narrow, open space in the heart of a dense and most noisome bazaar, containing a little Venice of open sewer cesspools. In fifteen years, as far as my knowledge as Principal went, two patients were attacked in the hospital of 300 beds—no attendant. In the adjoining college, one student was attacked, but no servant. This, in a city where cholera rages as a pestilence for four months in every year, and from which the disease is never absent. I look upon those three cholera cases merely as our share of suffering from the cholera influence which hung constantly over us throughout all those fifteen years.

I may note that, had we inoculated all our people, we could have boasted that the practice was triumphantly successful. Then, with regard to my own personal and home experience (which is also the experience of thousands of other well-to-do Englishmen, their wives and children, who lived in Bengal while I did)—in all those years, although we were certainly very scrupulous in choosing the best drinking-water, and in having that filtered and boiled, it cannot be doubted that, if there be a cholera poison—which, if it be a solid or a gas, must exist abundantly in the horrible dust (Budd) and fog of Calcutta—we must have taken it into our systems almost in everything that we swallowed and in every breath that we drew, to say nothing of the fact that, during more than half my long service in

India, we lived, with windows open day and night, less than a hundred yards to leeward of a cholera hospital.

All those who think as I do on this subject are constantly saying to the propagationists, 'You are too ready to read *cause and effect* where we merely recognise *striking coincidence*.'¹ Still, as the opinion that the disease is propagated by water containing cholera dejecta is held tenaciously, at this moment, as a guide in preventive legislation, by many physicians of great experience and scientific eminence, and as the question is one of exceedingly great importance to suffering humanity, I have no hesitation in stating my opinion that it ought, should cholera arise among us here, to be put to a crucial test '*in corporibus volorum*.' As this disease is not proved to be communicable to the lower animals, and as the anti-vivisectionists would probably not allow us to make experiments if serious doubt remained, the Government ought to be moved to permit it to be tried upon criminals condemned to death. As these poor creatures would be well attended to, I believe that, even if it be true that the dejecta contain and communicate the specific poison of cholera, at least three-fourths of their number, if not all, would escape with life.

Until this is done, and afterwards, I shall retain the belief that, on every ground, we ought, as far as possible, to avoid swallowing cholera matter, not because I consider that this is the specific poison of cholera, but because it is a putrid irritant which is likely 'to disagree,' and so to become an *exciting* cause of cholera, as Epsom salts and putrid fish unquestionably are.

The discovery of the cause of cholera will probably never be vouchsafed to a man of narrow and one-sided views. I believe that nothing valid will be revealed to us unless we grasp and correlate *all* proved facts. He who enters upon the quest must recognise no opponents. All working in the field must be regarded as fellow-labourers and helpers, save those who are absolutely ignorant or dishonest. He who is so happy as to grasp the truth will certainly perceive that it is reconcilable with all the good work of men holding all opinions. 'Why,' it may be said, 'you affect broad views, while you are really a bigoted anti-propagationist!' My reply is, 'Not so; I am an agnostic, with a strong anti-propagationist impression, which may possibly be removable, but which is quite unshaken at present.'

As I have already said, I have generally noticed that those of my most experienced brother officers who have been propagationists, have made their observations chiefly in the epidemic field of cholera

¹ Mr. Froude has recalled Luther's commentary upon astrology. 'It is like dice-throwing. You say that you have a pair of dice that always throw twice six—you throw two, three, four, five, six, and you take no notice. When twice six turns up, you think it proves your case. The astrologer is right once or twice, and boasts of his art. He overlooks his mistakes.'

in Upper India, while the anti-propagationists have generally practised in the endemic area of Bengal Proper—a very large proportion of those whose observations have been restricted to the cholera epidemics of Europe being propagationists: it therefore stands as a fair question, Is or is not epidemic cholera propagated by the dejecta, while the endemic disease of Lower Bengal is not? My mind has, for many years, been open to this question, which has often occupied my thoughts. But I have never seen or heard anything which, upon close investigation, shakes my firm impression that a specific cholera poison is not contained in the stools.

Pace the shade of M. Fauvel, I do not hold these views 'in accordance with the commercial interest of my country,'¹ being quite unaware what that 'interest' is—save that it appears to me that, if I were a Bristol merchant, it would not be to my 'interest' to see that port impested by cholera. It has never been my fortune to enjoy the rest afforded by a prolonged term of quarantine; but, having been conducted through the Alexandria railway station in five journeys to and from India, I can affirm that, adding together all the terms of my stay at that place, not three hours of my life have been spent on shore at Alexandria. I have been conveyed in what appeared to be a pig-boat to and from the steamer, and have been taken across the desert in a vehicle of bare boards which had the look and comfort of a sheep-van. I have seen an Egyptian receive a sixpenny piece from an Englishman in the bowl of a gravy-spoon filled with water; and I, together with the rest of my fellow-passengers, have been constrained by a straw-hatted official to soak and wring out, in public, the contents of my clothes-bag on the deck of a P. & O. steamer, in the shadow of Monte Cristo's prison—apparently with an intention of liberating the cholera germ, and of giving it a habitat in French waters. Still I remain absolutely unconvinced of the preventive efficacy of sanitary cordons and of quarantine in cutting off the approach of that which does not travel, and in arresting the propagation of that which is never propagated.

The following data—the validity of most of which is recognised by many, if not by all physicians of Indian experience—may be taken as applicable to our present threatened position in Europe. All of these conclusions are based upon many more facts than can be given here.

If a strong epidemic wave rises among us, its destructive effects will not be wholly avoidable. In India, good hygiene is generally a great protective. English officers and merchants, their wives and children, are comparatively rarely attacked, their good hygiene being their main safeguard; but, in two Indian epidemics, within my knowledge, the officers suffered greatly. So, in one of the worst

¹ Report of a controversy in the Paris Académie de Médecine (*Daily Telegraph* or August 6, 1883).

small-pox visitations which I watched in Calcutta, persons whose faces were seamed by a previous attack fell victims.

Whatever treatment may be adopted, a very large percentage of those first attacked will probably die. It is a fact noticed always and everywhere in India, that, at the first onset of an epidemic, and when an endemic first sets in, the loss of life among those attacked is terrible, the disease having an intensity which, in a majority of cases, defies all treatment. Later, when the outbreak is on the decline, there is usually a strong tendency to recovery. This law in the disease has repeatedly led inexperienced practitioners into a very transparent mistake. One who has recently come to the country frequently makes this observation: 'When cholera appeared, in February, I tried all the old remedies, but found them useless. Being almost in despair, in May, I gave album græcum in three-grain doses every quarter of an hour, and cured sixty per cent. of my patients!' The fallacy only becomes apparent, even to the discoverer himself, when he comes to employ his specific *at the commencement* of the next outbreak. This law receives some indirect illustration from the fact, given by Dr. Macpherson, that, in an analysis of 1,354 cases of cholera in Europeans in Calcutta, 'the rate of mortality during the hot or cholera months was 56·2 per cent., and 45·2 during the others.'

The following data, also given by Macpherson, are still more explicit:—In an outbreak at Kurrachee, in Scinde, of the first 100 admissions 79 died; second, 66; third, 50; fourth, 40.

In cholera outbreaks, he who enters the epidemic or endemic area encounters special danger. In India, no one can be considered safe on first entering a cholera-impested area. Thus, when I looked round my cholera ward, the native patients generally had much the appearance of Calcutta people; but, on inquiry, it was almost always found that the majority were outsiders who had not been many days in the city. It was hoped that the introduction of an excellent water supply would put an end to this, but it did not—in my time. All travellers, Europeans and natives, from up country incur this liability. It is a common saying among natives, that everyone who enters Calcutta is liable to bowel complaint;¹ and I am confident that no one who arrives in Calcutta from Europe can regard himself as being perfectly free from danger of an attack until he has passed through a cholera season. The Europeans who die of this disease in Calcutta are mostly sailors, lately arrived in port, and other new-comers. Hence the necessity for moderate care in living during the early months of residence in India.

¹ So also it was observed by a medical officer, long resident there previous to the Mutiny—I think Dr. John Balfour—that the water of Delhi was so bad that nearly every European who went there was attacked with some disorder of the bowels.

The works of Dr. John Macpherson, the 'Annals of Cholera' and 'Cholera in its Home,' which may, at the present crisis, be studied throughout with the greatest interest and profit by every medical man in this country, contain important illustrations of this law, among the most striking of which is the following¹:—

'A first-class ship arrived in the Hooghly in the early days of January. There was no cholera in the vessel coming up the river, or during the month after its arrival. There was very little in the city of Calcutta.' [Still, there is generally a brief cholera season at about that time in Calcutta.—N.C.] 'A party consisting of a gentleman and his wife, a European nurse, a young lady, and two children, landed from the ship in perfect health on a Sunday. They went to a house in which there had been no cholera for several years—not, however, in a good situation (Circular Road, opposite to Elysium-row) and small for the party that was received in it. After their arrival, there was a total of eight adults and ten children in the house. Of these, two adults and three children were residents, two adults and three children were arrivals from up-country, and the rest were the new arrivals. Five of the new arrivals occupied a small room on the upper flat—that is, three females and two children—they had one bathing-room off it. The rest of the occupants of the house were partly upstairs and partly down. The nurse and children appear to have had slight diarrhœa during the week, and the young lady ate on Saturday some indigestible food. The children, having slight diarrhœa, got a dose of Gregory's powder from the mother on Sunday morning. I was sent for at 8 P.M. of that day, and found one child far gone in cholera; she died at half-past twelve at night. The other child was in the earlier stage of the disease. About midnight, the father had an attack which was checked; and, towards morning, the nurse was seized. She was sent to the General Hospital, close by, at 7 A.M. and died at 7 P.M. All who could possibly leave the house (all but its resident occupants) were ordered out of the house. The young lady, who had been in devoted attendance on the children, went to Wilson's Hotel. There she was seized on Monday night, and died at 4 P.M. on Tuesday. The second child eventually recovered. One of the occupants of the room downstairs was at the time suffering from chronic diarrhœa, but he was not attacked. No more cases occurred in the house or in the servants' outhouses.'

Here, as Dr. Macpherson observes, the sufferers had just arrived from sea; and it may again be noteworthy that, almost precisely as happened in Mr. Macnamara's case, where of nineteen persons who swallowed water polluted by cholera excreta, five were attacked with cholera, so, of the eighteen inmates of this house, five were also attacked.

¹ *Cholera in its Home*, p. 36.

A very few days after the 19th Regiment arrived in Calcutta, early in the Mutiny, my friend, Professor Longmore, of Netley, took me to the bedside of an officer who had gained remarkable distinction in the Crimea, and was known as 'the boy Colonel.' Colonel R. landed his regiment about Christmas-time (when, as we have seen, there is always a tendency to a brief cholera outbreak). In his first arrangements for his men, he had great mental anxiety and bodily fatigue. He sank in a few hours in the collapse stage.

'*The incidence of cholera will always be heaviest upon the most insanitary localities.*' I have endeavoured to state this generally recognised law in the words used by that great sanitarian, Mr. Edwin Chadwick, at a meeting of the Epidemiological Society on July 4, 1883.

As I have already mentioned, well-to-do ladies and gentlemen, who have resided for more than a year in Calcutta, or elsewhere in India, are rarely attacked. When they are, it is usually found that they inhabit certain notorious spots—such as Lall Bazar and its vicinity (the unhealthy quarter most frequented by sailors), the old town around Tank-square, and the site of an old filled-up tank in Chowringhee. My friend, Dr. Thomas W. Wilson, kept in his study a map showing every house in the place, on which he marked the houses where cholera cases occurred within his experience. It was in such localities as the above that his black marks lay thickest. We inhabited for some months a house which, at first sight, appeared to be delightfully situated, but which I soon found to be surrounded with insanitary influences. When I left, on the occurrence of a case of grave remittent fever, I was told that the house was notorious for cholera; and I know that, in the course of a few years, two well-to-do Europeans died of cholera in one of its rooms.

Dr. Macpherson mentions¹ an instance which was known to every medical man in Calcutta. Many years ago, certain large palatial houses in Middleton-row were almost deserted, being considered a hot-bed of cholera. The site had been occupied by a large tank in Sir Elijah Impey's park, which had been filled in, doubtless after the old manner in Calcutta, with every kind of filth and refuse.

Next we have the well-established fact that *it is dangerous to travel in the cholera area*. Hence it is that pilgrims, who endure the most terrible exposure, privation, and fatigue, appear so prominently in the history of Indian cholera. The natives have a saying that the widow who goes upon a pilgrimage performs as great a sacrifice as she who is burnt on her husband's pyre.

Those who are well lodged and in comfortable circumstances have a great prospect of escape, if they remain at home. Well-to-do Europeans, resident in Calcutta, suffer very little from cholera, but travellers

¹ *Cholera in its Home*, p. 22.

undergo great peril of that disease, even when there is but little fatigue, privation, or exposure. A late venerable archdeacon, who had enjoyed good health for many years in the cholera atmosphere of Calcutta, went up country on visitation, and died of cholera at Ghazipur. The only member of our rather large European community who suffered when epidemic cholera visited Chittagong, in 1849, was the chaplain. He came in from Tipperah, when cholera was raging in my jail, ate a hearty dinner, went to bed, and was almost immediately attacked. His was a very severe and typical case, and recovery was difficult. The following case also came within my own notice. Two young married ladies arrived at Calcutta by the same ship in the cold season. One of them remained in Calcutta nearly three months, and then accompanied her husband from our house at Howrah, in high health and spirits, to the barracks at Chinsurah. This was early in the cholera season. Meeting the other lady in the neighbouring town of Hooghly, she accompanied her friend to church on the first Sunday after her arrival. Both were attacked with cholera on that day, and both died. A year or two subsequently to this, a lady, long resident at Chinsurah, came down on a visit to a relation living in one of the best-situated houses in Calcutta. She was immediately attacked with fatal cholera. This lady's husband subsequently left Chinsurah, and was stationed, for a short time, in Burmah. He died of cholera on his return to Calcutta. Such cases are known to every officer of Indian experience.

Shortly before the first appearance of this chapter, I heard of a case illustrating this law. A wealthy lady had made a tour through India, and, having come down to Calcutta from the Upper Provinces, invited a friend of mine, who called on her arrival, to luncheon on the following day. Previous to that time, she was fatally attacked by cholera.

In old times, within my recollection, occurrences like the following were frequent in India. A party of healthy European soldiers would be embarked early in the morning at Chinsurah on a well-found troop-boat, towed by a steamer. On arriving at Fort William, Calcutta, at midday, several men, dying of cholera, would have to be landed and taken to the General Hospital. Again, a vessel, conveying coolies to the Mauritius, would leave Calcutta with all on board healthy. Cholera almost inevitably appeared, with more or less severity, before the vessel passed the Sand Heads, 110 miles down at the mouth of the Hooghly, and ceased when she was once fairly at sea. When it was customary to send European soldiers up-country from Bengal on country boats—which, when not overcrowded (as they too often were) were comfortable and wholesome—the results were frequently most calamitous. An account of one of the most disastrous instances—that of H.M.'s 87th Royal Irish Fusiliers, in 1849—is detailed in my work 'On the Means of Preserving the Health of European Soldiers in India.'¹

¹ *I. A.*, No. 10, p. 706.

Between September and the following March, this fine regiment, 1,036 strong, lost, on their way up, from cholera, dysentery, and fever, 217, exclusive of 1 officer, and 29 women out of a strength of 73, and 29 children out of 103. In the above-mentioned work, are cited at length several instances illustrative of the law that native troops, when embarked on country boats, were almost invariably attacked with cholera, as in the case of the 8th Native Infantry, who, on the voyage from Cawnpore to Benares, at the end of August and beginning of September 1856, lost, in seventeen days, 42 men out of a strength of 1,115. Nearly all authorities agreed that these outbreaks of cholera, among Hindoo troops, were dependent mainly upon the use by the men of ill-cooked nutriment—such as *chobanee* (parched rice) and soaked gram (horse beans). I feel confident, however, that these parties merely fell under a law in cholera to which men of all habits and races are alike subject. Raw rice and beans could not *cause* cholera, they could only *excite* it; and we see that European soldiers, to whom these articles of native diet were unknown, suffered precisely as the natives did.

I had imagined until now that these calamities had become infrequent in recent times; but, as lately as April 28 of the present year, I find the announcement from the commissioner at Mandalay:—‘Cholera attacked 43rd Regt. on voyage up river; fourteen deaths, including three followers. Steamer halted.’

Here we come upon a concurrent law, the explanation of which has perplexed many. *Whenever cholera attacks a barrack or jail, the surest means of staying the pest is to remove those who are still well to a judiciously chosen camping-ground.* We did this when my jail at Chittagong was very severely attacked by epidemic cholera in 1849. Not a single case occurred after the prisoners were promptly removed to camp. Mackinnon gives an instance in which, cholera having broken out in a Queen’s regiment in Fort William, part of the wing of the regiment was moved to Chinsurah Barracks, with the best effect. Upon this interesting point Dr. John Murray’s paper ‘On Removal in Epidemic Cholera’¹ should be consulted. It is mainly due to native recognition of this law that so many ruined villages are noticed in travelling through the swampy districts of Lower Bengal; and that every large city, like Dacca, is observed to be environed by a wide belt of hut foundations.

Those European and Indian observers who contend that a cholera patient is a focus whence the disease will radiate by the admixture of cholera dejecta with drinking-water or with dust, urge that seamen and pilgrims, coming from cholera-impested places, convey the disease to healthy localities, having ‘followed the great routes of trade,’ which, they consider, cholera especially besets. This opinion is assailable on various grounds. Take the following. We know that

¹ *Transactions of the Epidemiological Society*, 1879-80.

the cholera endemic becomes very grave in Calcutta at about the middle of February. Let us, for the sake of argument, assume the possible case that epidemic cholera will begin to prevail in England at precisely the same time. John Smith, sailor, arrives from Alexandria, which is impested by cholera, at Southampton, or at any other English port, on February 12. Wherever he arrives, he will be said to have 'followed one of the great routes of trade,' as probably ships from Alexandria arrive every week at several English ports. His brother, Thomas Smith, sailor, arrives, also from Alexandria, on the same day, in the port of Calcutta. Both men drink and knock about on shore, are attacked with cholera, and die. On the following day, cholera breaks out at both ports. Now, we of Indian experience can trace perfectly the sequence of events in the case of Thomas. He also 'followed one of the great routes of trade.' But no Calcutta medical man will dream of asserting that he brought the endemic pestilence into that city. All will immediately agree in declaring that he fell a victim to the cholera poison, which he found there, just as a dog dies when he is thrown into the Grotto del Cane and is choked by its gas. Why does his death precede by a day or two any cholera death among the fixed inhabitants of Calcutta? He falls a victim to the law, illustrated by a multitude of facts like those of the cases of Colonel R——, and of the persons attacked in the Circular Road house (given at page 289), under which, in India, *the gravest and speediest incidence of a cholera outbreak is always upon those newly arrived in the cholera area*, especially if, as is generally the case, they be exhausted by fatigue, or be very imprudent, immediately upon arrival. Our opponents will unhesitatingly declare that John Smith brought the epidemic cholera from Egypt to England. We 'non-propagationists' will reply, as steadfastly, that it awaited his arrival in England, and that both men were attacked and died under one and the same law of disease.

In the February of any year, a Calcutta medical man, addressing the sailors newly arrived, would say, 'Some of you will be attacked with cholera before this month is out, unless you are unusually prudent and careful.' At a time like the present,¹ our port authorities ought to have all recently arrived sailors duly warned and instructed where they can, with the utmost ease and readiness, obtain medicine gratis, whenever they are attacked with bowel disorder.

Exciting Causes of Cholera.—Principal among these are *all causes tending to produce nervous depression and exhaustion*—want, excess, exposure, fatigue, panic—and *everything which, in popular language, 'disagrees' with the stomach*. A circumstance which almost invariably attends cholera, dysentery, and the gravest forms of diarrhoea

¹ This remark first appeared in September 1883. Now, in the middle of 1886, it is nearly equally applicable to our situation in Britain.

in India is that, immediately the morbid process sets in, the stomach signally fails in its power to digest, and that more or less suspension of digestive power frequently continues far into convalescence. Hence the fact that, in cases of subacute (commonly termed 'chronic') dysentery, when the patients are imprudent, as they mostly are, nearly every kind of undigested food may be looked for in the stools. The chaplain of Chittagong, whose attack I have already alluded to as having occurred soon after he had dined and gone to bed on returning from a journey, while epidemic cholera was raging in the station, vomited his plentiful meal of beef and potatoes entirely undigested, although there had been quite time enough for digestion. I carefully examined the vomit: meat and vegetable, which had been bolted hungrily in great masses, appeared quite unchanged and untouched by gastric juice. It appeared astonishing that keen appetite could have existed in such a condition of the system. We generally dine late in India, but those who are prudent *never eat fruit at night*. This is best taken at breakfast. All experience shows that Europeans are generally attacked with cholera at night or in the 'small hours,' and natives after a full meal (Macpherson).

I have already alluded to the fact that most of the inmates of my cholera ward in Calcutta were strangers, attacked shortly after their arrival in Calcutta. It was believed that their disease was excited by *drinking foul water* from filthy tanks, in their ignorance of better sources of supply. The majority of European cases in that institution were sailors lately arrived in port, who had been imprudent on landing, and who were frequently known to have drunk Hooghly water, taken up over the ship's side, which is often brackish, and which is always contaminated with every kind of city filth, probably including *cholera excreta*. I have already mentioned that an officer told me that he and a brother officer, having duty in an up-country bazaar, became so much heated and fatigued that they went into the shop of a native dealer, and asked for two pint bottles of ale. His companion emptied his at a draught. That which was poured out for my friend was so horribly decomposed that he rejected it, and asked the other how he could swallow such stuff. The reply was, 'I was so thirsty that I could have swallowed anything.' The poor fellow was attacked with cholera on his return to quarters, and died. (*Vide* p. 22, where a similar case is noted.) An administrative medical officer, when travelling from the plains to a hill station, suffered much from thirst and, unguardedly, drank milk not long after drinking beer. He was soon afterwards fatally attacked with cholera. A very healthy gentleman and his wife, friends of mine, were, during part of the Mutiny, shut up in the fort at Allahabad, the sanitary condition of which was then most evil, where they endured considerable privation. As the danger lessened, the refugees were removed to neighbouring places in

detachments. Mr. H—— and his wife were separated, and died of cholera in a few days, neither hearing of the other's fate. Here this disease, which prevailed sadly among the refugees, was attributed to too great freedom in eating meat, fruit, and vegetables, of which they obtained large supplies. One of the few cases of European ladies who were attacked with cholera in Calcutta during my long residence there, was a civilian's wife who lived in a distant suburb, Alipore, and whose seizure followed a rather fatiguing morning shopping in the town, after which mangoes were eaten freely.

I attended with a brother officer the fatal case of an English lady attacked during her first cholera season in Calcutta, after having breakfasted upon corned ox-tongue. A European man recovered, in my ward, from cholera, caused, as he believed, by eating corned beef. It was thought that the lower class of butchers employ arsenic in 'curing' tainted meat. *Strong purgatives, especially Epsom salts or other hydrogogue cathartic salines*, are so notorious as exciting causes of cholera, that I, throughout my career, never gave salts or seidlitz powder. According to Indian usage, *I never gave any purgative at night*. I was called by a medical man to visit his mother, who was attacked with cholera after taking a dose of sulphate of magnesia. One of my last fatal cases in the country was that of a poor European woman, in whom the attack was excited by a dose of well-known 'antibilious' pills.

Variation in the Types of Cholera.—It is needless in the present day, with the works of such observers as Macpherson, Goodeve, and Macnamara before us, to enter into a description of the common and unmistakable symptoms of malignant cholera—the stage of premonitory diarrhoea, when it is present; the stage of algide collapse; and the stage of reaction, with its perils from arrest of hepatic and renal excretion. As I emphatically observed of Indian fevers that *the type changes incessantly*, so it is with cholera. I always noticed a distinctly marked variation, not only in the type of each outbreak, but also in the condition of each patient—every man's case has its own distinct individuality. Consequently, no disease stands more in need than cholera does of special treatment according to the peculiarities and exigencies of every case.

This is a law which every observant man will be able to read clearly soon after the disease comes before him, and which is laid down by Dr. Macpherson in the following pithy words:—'In all essentials the disease is the same as when it first broke out'; but then 'all observers are agreed that the cholera of one season varies from that of another, just as the character of fever changes. Some of the most striking variations are the degree of blueness of the skin, the early occurrence of collapse, the amount of vomiting and purging, or of cramps, the frequency of consecutive fever, the degree in which the

disease is amenable to treatment.' To these variations may be added an appearance of bile or blood in the stools, great differences in the condition of the mucous membrane and follicles of the ileum, especially as regards vascularity and exudation, a tendency to the formation of ante-mortem clots in the right heart. In one outbreak, there will be a prevalence, as we begin to hope that our native patients are safe, of sloughing of the cornea, which first becomes dull and sunken, evidently from arrest of nutrition. On another occasion, there will be a tendency, also in natives, to sloughing of the scrotum. We cannot, at first, judge whether the occurrence of the stage of reaction will be early or late. In one autumnal outbreak, some of my patients remained collapsed for three days. The tendency to serious head-complication in the stage of consecutive fever varies greatly; so also does the disposition to retention of the first urine when the bladder is full. Cholera spasm or cramp is not very common or excessive in the weak-muscled natives of Lower Bengal (who endure tetanus much better and longer than Europeans generally do) or in women. I have often thought that the fatal result was determined, in strong European sailors, by the severity of the cramps. I had two sailors lying side by side—one a perfect picture of healthy athletic muscularity; the other sickly-looking and meagre. I could only account for the death of the more powerful man and the recovery of his comrade upon this ground. I did not see, in Bengal, that muscle-tearing cramp which has occurred in England.

I cannot quite understand with what view the authorities, in 1840, added to the Bengal Medical Return the disease *Cholera Biliosa*. By this they may have intended to designate a form of malignant cholera in which the stools are bile-tinged. I saw one of these fatal cases in Calcutta, and heard of another—the two examples occurring more than twenty years apart. Dr. Morehead found these cases rare. I do not think, however, that it was intended to set apart this very exceptional form of the disease, because, I repeat, it is, self-evidently, true algide cholera wherever it occurs.

The separate heading was, doubtless, intended for those cases of violent—but, in my experience, always safe and transient—*Bilious Flux*, perhaps attended with vomiting, which are frequent among Europeans at the end of the cold weather, just as the cholera season is setting in. The liver, having been rather congested during the cold weather, suddenly relieves itself by an enormous flow of bile, which produces considerable prostration, and still more alarm. The first case I saw was treated by a native sub-assistant-surgeon, who gave a scruple of calomel and the same quantity of soda. After taking this, the patient went to sleep, and was nearly well in the morning. I used to give the soda without the calomel—indeed, the disease relieves itself. I noticed that one of these thorough purgations generally left a peculiarly clean tongue, not raw, but perfectly moist and healthy. Many,

when I first went to India, called this 'bilious cholera,' and I suspect that many of these cases—all of which were 'cured'—were included under the head of 'cholera' in the old returns, previous to 1840, with serious detriment to the accuracy of their cholera death-rate.

Cholera is often, but not always, ushered in by *Premonitory Diarrhœa*. This can generally be arrested and an attack of cholera prevented by the timely use of opium. Wherever cholera was prevalent in an out-district, we used to send to all the police *thannas* large stocks of 'cholera pills' of which my friend Dr. Waring gives the prescription in his excellent work on 'Bazaar Medicines'—opium, black pepper, and assafoetida, of each twenty grains; beat them well together and divide into twelve pills: of these one was the dose, repeated every hour if required. With a view to speedy solution, we used to give these broken up in a table-spoonful of brandy and water. These pills used to be in high repute. I generally gave twenty minims of laudanum in a draught with chloric ether, sesquicarbonate of soda, chalk mixture, and cinnamon water. Omum water, distilled from the seeds of carum (ptychotis) ajowan—(Waring)—is much used as a carminative vehicle in the Madras Presidency.

Cholera simplex is readily distinguishable in a country like England, where malignant cholera does not appear except as an epidemic, save when a not unquestionable sporadic case occurs once or twice in a lifetime; but in Bengal Proper, where true cholera is constantly endemic, it is sometimes hard to differentiate the two maladies save upon a retrospective view. We are told that a patient is dying of cholera, and we find him with or without vomiting, and passing thin, copious, and almost colourless stools. He is greatly alarmed, nearly algid, and very low; has a thread-like pulse, and complains of spasms. At first sight, this may be a case of cholera. We give a dose of opium—the patient sleeps, and does well. It is noticed in these cases that there are never the characteristic rice-water evacuations of cholera. The radial pulse is not arrested for hours, there is never true collapse or complete arrest of urine. An elderly man, of great scientific eminence, retired from long and very arduous service in India, suffered from most intractable chronic white diarrhœa, which was probably true hill diarrhœa. He remained for upwards of two years and a half in a healthy midland English town, very slowly but decidedly losing strength. He was certainly not careful as regarded his diet, and he travelled moderate distances by rail a good deal. In his second summer at home, the weather being very hot, I received a telegram saying that he was dangerously ill. I could not reach his house until after midnight. He was lying on his side, as those about him thought in calm sleep, but was dead. I was convinced, upon inquiry, that he had been attacked with English cholera, under which his exhausted powers sank.

Persons subject to chronic diarrhœa and subacute ('chronic') dysentery, and those who are said to have 'irritable bowels,' are especially liable to be attacked with cholera.

'The History of Cholera, in India and Elsewhere,' has found a most able illustrator in my friend Dr. John Macpherson.¹ Here it will be sufficient to mention that its first great epidemic outbreak, in modern times, commenced at Jessore, in Lower Bengal, in the year 1817, which, as I have already shown (p. 94), was a season of singular zymotic activity throughout the peninsula of India. Thenceforward, it has been constantly endemic in the Gangetic Delta; rarely, but distinctly, appearing as an epidemic in that locality, as in 1849. As I have shown in a previous work,² the endemic cholera of Calcutta is most severe in the hot season, which extends from about the middle of February until the commencement of the rains (about June 20). But the statistical data which I have there cited show that the disease is very destructive throughout the year in Lower Bengal. I believe that Calcutta never has a day unmarked by a cholera death. Now and then, there is a rather severe outbreak at about the close of the rains, and another very marked but less heavy one at Christmas time. In the North-Western Provinces, cholera begins to prevail when it is on the decline in Bengal. In the former, the disease prevails most in June, July, August, and September—July and August being the worst months. In the North-West Provinces, the disease can scarcely be said to be endemic. There, a severe epidemic is to be looked for about once in three years.

It is well known that the late Dr. James Lumsdaine Bryden devoted many years of most careful observation to the investigation of the habits of Indian cholera. In pursuing his inquiry, he had the singular advantage of being able to trace, in the office of the Sanitary Commissioner, every movement made by cholera in India during the lengthened period of his quest; and there cannot be a doubt that one placed as he was, with the whole of the past history of the movements of epidemic cholera tabulated before him, and in constant receipt of official intimation of every outbreak of the disease throughout the wide field of his supervision, would be able, as he was, to predicate with considerable accuracy the probability of the appearance of the disease, at a stated period, in any given locality, and thus to afford valid advice with regard to timing the movement of troops, the necessity for special sanitary precautions, &c.

Bryden's views, as far as he had endeavoured to lay them before the profession when his valued life was brought to a premature close, may be studied in the annual reports of the Sanitary Commissioner with the Government of India; in a Blue Book by Deputy Surgeon-

¹ *Annals of Cholera.*

² *Means of Preserving the Health of European Soldiers in India.*

General Dr. Jeffery A. Marston, 1878, Appendix No. 12, page 300 ; and in his own ' Suggestions for the Systematic Study of the History and Relations of Cholera,' in the ' I. M. G.' for October 1, 1886.

Next to fever, cholera is the most destructive of all Indian diseases. Sir Joseph Fayrer shows that, in the year 1879, there were 270,552 deaths from this cause, out of 4,975,042 registered deaths from all causes.

I have said that, in Lower Bengal, the great endemic season is from the beginning of the hot weather in February until the setting-in of the rains in the third week of June. There is generally stormy weather at about the end of the second week of March ; this almost always sends in a batch of cholera cases, *palkee* (palanquin) bearers and other poor creatures whom the rough weather has suddenly chilled ; then there are a few days of cool weather, in which there are very few cholera admissions ; and then great heat sets in, and with it come a crowd of cholera patients. My cholera ward held twelve beds, and the cases usually came in so fast during the epidemic season that it occurred to me nearly every year that I should have to find extra accommodation ; but the necessity for this always happened to be prevented by the rapidity with which patients died and left beds vacant.

When, during the very hottest weather, and when the prevalence of cholera is at its height, a storm cools the atmosphere, it, as I have already said, chills and endangers those who are most exposed ; but such a change almost certainly abates, for a little time, the severity of the outbreak. Thus, I have before me a report in which it is stated that, not very long previously,¹ the population of Manilla was being decimated by cholera, when at the end of October (1882 ?) a tremendous hurricane swept over the island, almost entirely destroying the town. In less than an hour from the commencement of the storm, not a single native house was left standing. But, on the following day, not a solitary case of cholera occurred, and not one had been reported since. Here we inquire—How long since ? In Calcutta, the *chota bursat* (little rains) occur at the height of the cholera season, at about the beginning of the last week in May. They influence the endemic in the manner which I have described ; but, during the twenty days of intense heat which generally follow them, cholera is very prevalent. When the rainy season sets in steadily, about June 20, we almost invariably have reason to consider that ' the cholera season ' is over.

With regard to *Measures of Prevention*, I am in accord with the believers in the communicability of cholera, in maintaining that, on the arrival of cholera cases in ships, they ought to be treated apart. But I would do this not as a sanitary precaution, but as a means of testing the question—did these people bring cholera into the country ? I think it is certain that the segregation of those members of a household who may be attacked ought to be strongly recommended, but

¹ I had not the precise date when this was first written in 1883.

ought not to be made compulsory when the other members of the family are energetic and courageous, or when due assistance is obtainable. When such removal is voluntary, it is advisable, not as a means of preventing the spread of cholera, but upon the consideration that a well-managed cholera hospital is the best place; as, there, due attendance is available night and day. In a private house, the services of four persons—a day and a night nurse, fully instructed, and two strong men to apply friction—would be absolutely needful.¹ Soiled clothing and bedding should be burnt, not with the idea that they are *fomites* of contagion, but because they are hopelessly nasty. Cholera excreta should be promptly removed and buried—for the same reason. The very best drinking-water, procurable at any cost, should be filtered and boiled and drunk as weak tea, not because we have to fear the presence in it of a cholera germ, but because bad and dirty water is a powerful *exciting* cause of cholera. So are all purgative and aperient medicines, especially salines. At most, two drachms of castor oil with the same quantity of compound tincture of rhubarb, in cinnamon water—*taken in the morning*—will suffice. Fruits and jams containing fruit seeds should be avoided. So also should potatoes, which are certainly hurtful, almost poisonous, in dysentery. The diet should be plain, but very good. Roast and boiled and broiled fresh beef, mutton, and chicken—nothing corned or salted or smoked; well-cooked cruciferous vegetables; stale white bread (home-made if possible) and really genuine fresh butter; cold tea or, in great moderation, weak spirit and water. All the generally accepted rules of good sanitation, civic, household, and personal, should be observed with absolute strictness.

Every member of the community may be bold in the confidence that his chances of suffering from the disease are small; and are only, if he be courageous and prudent, those which he shares with everyone else; and that his danger is not, in the very least, increased by the occurrence of cases in his household, or by visitation or attendance upon the sick.

Treatment of Cholera.

The states of the liver and kidneys in cholera are so full of interest and importance pathologically, and still more in a therapeutic

¹ A European officer, attacked with cholera, had, what is very unusual in India, three adult and active ladies constant in their attendance. I said, 'If each of you will alone attend him for a given time, you will be most useful; but, if you all continue to surround his bed night and day, you will all break down in a few hours.' They positively would continue to attend in their own manner, and all speedily failed. One of the chief uses of a cholera hospital is the service afforded to the sick by its competent body of resolute trained attendants.

point of view, that it is surprising that they have never yet been made the subjects of thorough clinical investigation. Algide cholera, being more remarkable than any disease with which we are acquainted for the almost entire absence of *χολή*, bile, from the evacuations during its worst stage, that of collapse, we should approach more clearly to descriptive precision if we called it acholia, and to pathological accuracy if we termed it Asiatic Pernicious Fever—thus wholly separating it in our ideas, as it is absolutely distinct in reality, from several disorders of the bowels with which it is still unhappily confused.¹

As a general rule, exceptions to which are rare, bile pigment is not positively detectable in the stools and vomited matter of the algide stage, during which it appears that the excretive functions of the liver are, in the large majority of cases, arrested as absolutely as those of the kidneys are in all cases. Dr. Edward Goodeve found that 'nitric acid occasionally gives red reaction in the' [rice-water] 'liquid. This is probably due to a small quantity of bile mixed somehow with the evacuations, but it is not certain what it is.' It is here to be remarked that any bile which may appear in vomited matter and alvine evacuations, whether during the collapse stage or early in the stage of reaction, must not be viewed as positive evidence that hepatic action is not absolutely arrested, as its source may be the gall-bladder. Tanner says that cholera stools contain only a trace of biliary matter. During the epidemic of 1868, Dr. Thudichum observed, in death during the stage of collapse, that the secretion of bile was completely arrested; and that, in extreme cases, a clear white fluid percolates through the hepatic ducts, free from bile, colouring matter, and albumen. It seems to be simply water, with a trace of alkali and a vestige of mucus. In some instances, the fluid was coloured, but contained no bile-acids. Placing cases of acute bilious diarrhœa or hepatic flux entirely aside, cases do occasionally occur in which the vomited fluid and the dejections, during the algide stage, are distinctly coloured by bile. This happened in the case of an administrative medical officer who died in Fort William, Calcutta, about twenty-nine years ago; and again, in 1874, among the constantly varying types of true cholera which occurred in Calcutta, there was one in which the fluids ejected during collapse were distinctly coloured by bile. In March, 1875, I had, in my ward, a European woman who was attacked with Asiatic cholera after having taken a well-known 'antibilious' purgative on the night of February 27. She died on March 4, at about eleven o'clock A.M., never having rallied from the collapse stage, and never having passed urine. She continued, up to two o'clock on March 3,

¹ The active mischievousness of this antiquated name is displayed in the fact that even so modern a writer as Dunglison says that it is derived from *χολή*, bile, and *ῥέω*, to flow, because it arises principally from a superabundance of acrid bile. This, in true Asiatic cholera, is, of course, precisely the reverse of the fact.

to vomit a bilious fluid, the quantity of bile voided being far too large to allow the suspicion that it came merely from the gall-bladder. Stools ceased on the afternoon of the 1st. They were from six to seven in the twenty-four hours, and were markedly coloured by bile. The severity of this case was much aggravated, and the evacuating treatment interfered with by a tendency to abort, the poor woman being in the second month of pregnancy. In 1874, there was a great and very unusual tendency to bilious vomiting in the reaction stage and at the beginning of convalescence from true cholera.

The ordinary and most favourable type of alvine evacuation, after reaction has become established, is a free and perfectly fluid fæculent and very bilious stool, of a warm brown colour, of which there should be some five or six during the twenty-four hours. Not unfrequently, the first evidence of improvement is the passing of what are locally known as 'Calcutta mud' stools, from their close resemblance to the dirty greyish mud of the Hooghly river.

The behaviour of the kidneys in cholera is, of course, far more open to clinical observation than that of the liver is. As a general rule, there is a considerable excess of bile-pigment in the first urine passed in cholera, and, even far on in convalescence, traces of biliary matter are frequently present in this excretion.

It is almost needless to say that, exceedingly as the types of cholera vary from year to year, and even from week to week, there is, in every case, complete suppression of urine during the algide stage; and that, however distinct the subsequent reaction may be, *we are never satisfied that the danger of collapse is over until urine has been passed*; and that we can never feel confident that our patient's second great peril, *the danger of uræmia, is over until the free excretion of perfectly healthy urine is re-established*.

Dr. Begbie found that the first urine is of diminished density, and generally contains albumen and bilious colouring matter, with the ordinary salts, but little or no urea.

In his article on Cholera, in 'Reynolds's System of Medicine,' Dr. Goodeve wrote: 'At first it' [the urine] 'comes scantily, high-coloured, and an ounce or two, with a strong and peculiar animal smell, deficient in urea perhaps, but not in all animal principles, generally albuminous, with many transparent casts. The albumen or allied compound, when present, is not always detected by nitric acid, though often by heat, giving a deposit not dissolved by nitric acid when the nitric acid test alone failed to detect it. The urine often turns pinkish with nitric acid.'¹

¹ The latest analyses of the excreta in cholera which I have seen are those by M. G. Pouchet (*Compt. rend.*, 100, pp. 220 and 362; *Jour. of Chem. Soc.*, London, May 1855; and *I. M. G.*, January 1866). He finds that the *Bile*, collected very shortly after death, is colourless and of a gelatinous consistency, and contains a

In the cholera season, extending from February to June 1868, I, with the aid of my very able house-physician, Baboo Raj Mohun Banerjee, commenced a series of observations upon the *Specific Gravity* of cholera urine, which, with an interruption of twenty months, I continued until I left India at the end of March 1876. The general result of very numerous observations was that the first urine usually owes its high colour chiefly to the presence of bile-pigment. Blood has not been detected. The fluid is turbid. *The specific gravity is generally high, from 1020 to 1026.* The reaction is generally acid. There is commonly a sediment of epithelium desquamated from the renal tubes. Albumen is probably always present, most frequently in small quantity. Such urine may be passed (the catheter has to be used once or twice in many cases) twice or thrice, generally with a high, but reduced, specific gravity, as 1019. The case advancing in progress to another very distinct stage, the flow of urine is more free, the fluid is usually transparent and pale, and *the specific gravity is low—from 1012 even down to 1002.* Bile-pigment is generally present, and may frequently be detected up to the time of the patient's discharge; it usually outstays the albumen. When the case does well, the albumen may be expected to disappear in from one to three days, but I have found it as late as the twelfth day. Renal epithelium has been noticed as late as the thirteenth day. During tardy convalescence, phosphates often appear in the urine. *Whenever, in the reaction stage of cholera, there are albumen and bile-pigment in urine of a very low specific gravity, what is now generally called uræmia, or, as it appears more correct to term it, cholo-uræmia, is to be apprehended and resisted.*

large proportion of water. The greater part of the solid matter consists of albumin and mucin, but leucine, tyrosine, and glucose are always present, together with fat globules, crystals of fatty acid, and cholesterin. The bile also contains products of the decomposition of the bile pigments, which oxidise and reproduce the colour when exposed to the air after removal of the albuminoids. Here I must remark that all examinations of the contents of the gall bladder in cholera ought to be made (1) when death has occurred in the collapse stage after free and long-continued colourless rice-water vomiting and purging, (2) in deaths happening early, and (3) late in the reaction stage. Even then, it may be doubted whether the gall bladder has not contained bile from the first. We know that the solids found in the first urine are unquestionably quite recent. The *urine*, during the period of reaction, we are told 'contains a larger proportion of organic matter, especially urea, and a smaller proportion of inorganic salts.' (This observation follows the statement that the very watery and almost colourless *stools* contain a relatively large proportion of urea and sodium chloride, especially the latter.) 'Sulphates are present in somewhat higher proportion with respect to the amount of urea, but the quantity of sulphur eliminated in the form of sulpho-acid is very small, and in many cases *nil*. The quantity of sodium chloride present is only one-tenth of the normal amount, and the proportion of phosphates, especially earthy phosphates, is also diminished. Among substances not normally present, the urine contains bile salts in variable quantity, albumen in somewhat large proportion (5-9 gram, per litre), glucose, often in very small quantity, and a peculiar albuminoid.'

The following abstracts of cases, taken from many, fairly illustrate these principles :—

Charles S., admitted collapsed March 6, 1868, at noon. Reaction commenced in forty-six minutes.

First urine passed on the morning of the 7th—scanty, high-coloured, specific gravity 1024, traces of albumen.

8th.—Bowels free ; no vomiting ; urine free, specific gravity 1012 ; no head symptoms.

9th.—Vomited once ; two stools ; *urine copious*, specific gravity 1002, traces of albumen. Towards the evening evidences of cholo-uræmia appeared, he became delirious and restless, was constipated, and vomited several times.

10th.—Specific gravity of urine 1020, traces of albumen and phosphates, bile-pigment. The head symptoms passed off and vomiting ceased.

11th.—Doing well.

17th.—Discharged.

Charles M., admitted in a state of collapse July 19, 1868. Reaction took place on the following day.

The first urine was passed on the 21st, specific gravity 1021, scanty, turbid, acid.

22nd.—Urine profuse, limpid, specific gravity 1004, traces of albumen. There were now symptoms of cholo-uræmia—dulness, drowsiness, constipation, and vomiting. Gentle purgation, sinapisms, and fomentations over the loins.

23rd.—Bowels moved several times ; vomiting ceased ; urine free, specific gravity 1008, traces of albumen ; no head symptoms.

24th.—Urine contained traces of albumen, specific gravity 1017. In other respects doing well.

25th.—Traces of albumen, specific gravity 1014.

26th.—Doing well. No albumen in the urine, specific gravity 1020.

29th.—Discharged, well.

Shiboo, admitted collapsed November 25. Reaction towards evening.

26th.—No urine ; head symptoms, dulness, vomiting, constipation, laboured respiration, with thickly coated tongue and congested eyes ; bowels constipated. Conjee-water injection ; mustard poultice to loins.

27th.—No urine ; uræmic symptoms ; purgatives, dry cupping and fomentation over the loins.

28th.—Urine scanty, specific gravity 1026, slightly albuminous, and with bile-pigment.

30th.—Urine 1004, slightly albuminous; head symptoms, vomiting, constipation, gentle purgation.

December 1.—Urine free, specific gravity 1012, albuminous; bowels open; no head symptoms.

2nd.—Urine specific gravity 1016, traces of albumen. Doing well.

3rd.—Urine 1016, no albumen. Doing well.

4th.—Urine free, 1018, no albumen.

5th.—Urine free, specific gravity 1022. Doing well.

9th.—Discharged, well.

The following case is singularly remarkable for the extremely low specific gravity of the urine, which continued far into convalescence. Up to the tenth day the urine was, as is usual, of high specific gravity; thenceforward, up to the time at which he was discharged, apparently quite well, on the forty-first day, its specific gravity did not rise higher than 1012 (once). Moderate, but marked, symptoms of cholo-uræmia were observed—drowsiness on the eleventh day; restlessness (jactitation) on the third day; and insomnia on the fourth and sixteenth nights; and severe headache on the thirty-eighth day, when the urine had the (in the late stage of this case) unusually high specific gravity of 1011.

The continued presence of albumen in the urine up to the twelfth day, of epithelium up to the thirteenth, and of bile up to the date of his discharge on the forty-first day, taken together with these symptoms, kept up constant apprehension of cholo-uræmia.

James W., admitted March 30, 1874, in a state of collapse. Reaction took place on the 31st, and urine was passed on this day, specific gravity 1022, without sediment, but with albumen and bile, bilious vomiting, and purging.

April 1.—Bladder relieved by catheter of twenty-nine ounces of urine of acid reaction, albumen about one-sixth, and bile present, no sediment, specific gravity 1019.

During the next six days, he made water freely; stools loose.

On April 8, (tenth day) the specific gravity was 1020, acid, with slight traces of albumen.

On the 9th, (eleventh day of illness) there was a tendency to vomit; there were five brownish-yellow stools. The urine was 1010, slightly albuminous, and it is noted that he was somewhat drowsy.

On the 10th, the urine was of the same specific gravity, with a slight trace of albumen.

11th.—Specific gravity 1012, no albumen.

13th.—Specific gravity 1006, reaction alkaline.

The urine continued to be alkaline until April 21 (twenty-third day), when it became slightly acid, with a specific gravity of 1011.

Thenceforward, the reaction continued to be acid.

The stools were frequent and thin up to the fourteenth day. On

the sixteenth day, there were only two semi-solid stools and no vomiting, and the specific gravity of the urine was as low as 1004; insomnia was complained of. On the seventeenth day, there was only one stool, no vomiting, the specific gravity of the urine was still 1004; the patient had slept well. On the eighteenth day, there had been a stool in the night, and there was another late in the day; the specific gravity of the urine was only 1002. On the following day also, there were two stools; no vomiting; he had slept well; the specific gravity of the urine was 1004. On the seventeen succeeding days on which the urine was examined, the range of specific gravity was 1010, 1009, 1010, 1006, 1010, 1007, 1005, 1006, 1008, 1010, 1011, 1010, 1009, 1010, 1010, 1011, 1008. During the whole of this latter period, the rule was one stool in the twenty-four hours, and the appetite was generally good.

This case shows that, while a very low specific gravity of the urine after cholera is always to be viewed as an indication for extreme watchfulness of the patient's condition, and for caution in treatment and dieting, it is not by any means invariably, as regards prognosis, an extremely grave sign. Still it will be noticed that, on the tenth day of the illness, there was urine of the specific gravity of 1020, with slight traces of albumen; on the following day, the urine being still albuminous, the specific gravity suddenly fell to 1010, and drowsiness was observed; the bowels were, however, free. Again, when, on the sixteenth day, there were only two semi-solid stools without vomiting, the specific gravity of the urine being so low as 1004, there was insomnia.

When I first assumed my charge at the Medical College Hospital, in the cholera season of 1862, I found that my colleagues had an established system of treating the collapse stage of cholera. Their leading objects were:—

1. *To arrest Vomiting and Purging.*—The rice-water evacuations were regarded as being, potentially, hæmorrhage. This was to be checked by styptics, principally acetate of lead; but opium and other narcotics were to be avoided as tending to produce uræmia, in the reaction stage, by locking up the excretions.

2. *To obtain Reaction.*—Diffusible stimulants, especially aromatic spirit of ammonia, were given steadily every quarter to half an hour until reaction set in. The body and limbs were rubbed with dry ginger powder by relays of active ward coolies—one to each limb. Large sinapisms were applied to the præcordial region and abdomen.

I then adopted and generally adhered steadily (except when new plans of treatment, which appeared to deserve trial, failed in my hands) to my colleagues' therapeutic *principles* in treating the collapse stage; but I made some changes in the *details* of their treatment. In

choosing a styptic, I preferred tannic acid (ten grains after the first, and five grains after every subsequent rice-water evacuation, whether by stool or vomiting) to acetate of lead, which, if absorbed, was likely to act as a depressant. To ammonia I objected on account of its affinity to urea, and I always preferred chloric ether as a diffusible stimulant. I ordered dry ginger friction only to the *trunk* of the body, considering that rubbing the extremities would tend to increase the already excessive congestion of the vessels of the great cavities. I, therefore, had eight hot-water bottles, frequently changed, in bags of thick flannel, applied to the limbs and trunk in every case, and had recourse to strong shampooing (kneading) of the limbs only when cramp was present.

Sydenham used *opium* in cholera, and specially thanked Providence for the gift; but Copland tells us that Frank and Schmidtman justly acknowledged the importance of Sydenham's observation that, when opium 'is given too early, much disorder of the bowels and abdominal organs, with more or less fever, continues afterwards to be complained of, evidently owing to the arrest of a salutary effort, and the retention of morbid secretions.' How, then, would these authorities have explained the undoubted good which opium effects in absolutely arresting the premonitory diarrhœa? Macpherson gives opium in the premonitory diarrhœa, and considers that, in the invasion of cholera, opium (laudanum) 'is still our chief remedy for a time.' When collapse is accompanied with a tendency to stupor, violent vomiting and purging having ceased, he takes it for granted that opium will have been given up. I certainly did not give opium where vomiting and purging had ceased, and where stupor was threatened. Macnamara gives opium in the early stages but not in collapse, even when frequent purging and vomiting continue. Drs. Aitken and Fergus maintain that opium is only to be given 'if the evacuations are still bilious, the pulse fair, and the skin warm,' and insist that, 'when vomiting, rice-water purging, and cramp set in, it is then too late for opium.' In my hospital practice, I scarcely ever saw a case of cholera which had not advanced at least to this latter stage; and it was then that, in respectful non-concurrence with authority, I gave opium. I long eschewed its use, except in premonitory diarrhœa; but, as my views regarding cholo-uræmia became clear, I gave laudanum a cautious trial in the collapse stage; and, in after years, generally used it. I think that it aids the tannic acid in arresting the gastric and alvine evacuations, every one of which reduces the patient's vital powers distinctly a step lower. It aids the stimulant action of the chloric ether; and, if it does, as I believe it does, assist our first therapeutic triumph, the establishment of reaction, we have no reason whatever to assume that it interferes with the restoration of the functions of the liver and

kidneys. Even if it be assumed that it does so, there lies before us the necessity of bringing about reaction, and we have at our command a clear line of treatment for the cholo-uræmia.

The patients were well watched, the laudanum was given cautiously, and was not continued after the rice-water evacuations had ceased. Thus used, I never saw it cause stupor or any other evil effect.

Although there may be no great power of absorbing drugs into the system during the algide stage, astringents, stimulants, and opium certainly do appear to act. It has been observed that, in most great outbreaks of pestilence, persons die suddenly almost without symptoms. They fall, perhaps in the streets, stricken down and overwhelmed by the concentrated intensity of the poison. I know of cases of cholera in which death occurred without vomiting, after one or two gelatinous stools. At the commencement of cholera outbreaks, when the natural tendency to death is at its maximum, we shall, I fear, always lose patients in the stage of collapse, but many patients are brought out of a state of pulseless collapse by the treatment described above.

We have shown that the first urine passed after an attack of Asiatic cholera is generally acid and turbid from inflammatory products or organic *débris*, and contains albumen and bile-elements. Its specific gravity is high, apparently only in consequence of the presence of the above products.

As the urine becomes limpid, its solid matters as well as the albumen and bile-elements diminish, and the specific gravity is almost always low. When it is slow in losing its albumen, and remains of a specific gravity between 1002 and 1010, and there is a tendency to constipation, cholo-uræmic danger must be apprehended, and vigilantly and actively guarded and fought against.

That dangerous complication which, setting in rather late in the algide stage, or almost at any period before the excretive functions of the kidneys and liver have become re-established, destroys multitudes of cholera patients by blood-poisoning (constituting the second and last great peril of cholera, collapse being the first) is generally called *uræmia*. To be understood etiologically and to be treated with success, it must, however, be recognised and dealt with as *cholo-uræmia*, because, in it, we have, in the bile-pigmented albuminous urine of very low specific gravity, and also in the absence of the free bilious stools which latter are essential to recovery from cholera, evidences that the terrible condition of blood-poisoning, with which we have to grapple, is due to failure equally of hepatic and renal elimination.

When the renal and hepatic tissues have been previously healthy, the uræmia and cholæmia of cholera are attributable, first, to congestion of the kidneys and liver, and, secondly, to lack of fluid in the system. Cholo-uræmia can be best guarded against by the use of large and repeated sinapisms over the liver and kidneys, by dry-cupping

over the kidneys; the steady application of a pillow-case half-filled with hot dry bran, in which the patient lies; large hot linseed-meal or *soojee* cataplasms to follow the sinapisms over the liver and loins; and the free use of *nature's own diuretics*, water and milk. Dr. Good-eve says 'water is the best diuretic.' After cholera, I have never dared to irritate the kidneys by more stimulating direct diuretics. These organs can, at this most critical period, only be *solicited* by the use of bland demulcent fluids, supplying the place of that which the cholera flux has almost completely drained the system of. Attempts to *compel* them to act can only tend to produce arterial congestion and uræmia. Still, not very long ago, there were some otherwise judicious practitioners who, becoming impatient at the slowness with which the congested serum-exuding, desquamating kidneys begin to act, while yet unsupplied with that water without which urine cannot be made, were unable to refrain from goading these already sufficiently over-burthened organs into premature action. One thought that 'a few doses of benzoate of ammonia' will act as a gentle diuretic—as if the kidneys were not already sufficiently troubled in a struggle to void urea! Another used 'solutions of the chlorate of potash and the like'; and a third was only satisfied when he added fire to fire by administering the tincture of cantharides!

In his remarks upon the treatment of the diarrhoea which follows cholera, a modern writer tells us that—'In those cases that are connected with defective secretion of urea, turpentine, either by the mouth or applied externally, is very useful.' We are not surprised to find that he adds: 'Cases of this nature are very obstinate, often continuing for months, and generally requiring change of air to the seaside, or a long sea-voyage to complete the cure.' This recalls the case of congestion of the posterior part of the liver, which I have cited in a previous chapter, in which we are told that, *although* the patient was bled three times to deliquium, recovery was tardy!

In treating cholera, I have always, on chemical grounds, avoided the use of ammonia in any form; have, in consideration for the state of the kidneys, interdicted the use of cantharides blisters, and have even refrained from the use of turpentine stupes.

Another ground for caution in treating the renal complication is the alleged fact that cholera is apt to lay the foundation of kidney (and may we not suggest of hepatic?) disease. Upon this point I can say little. In civil practice, we have very few opportunities of tracing our patients' after-history. Officers of the A.M.D. might frequently watch men recovered from cholera for long terms, noting their habits of life, and examining the urine from time to time. I have known people who had suffered from cholera who appeared to enjoy fair health for years. In one of my voyages, a P. & O. Co.'s steward came up to me and said that I had brought him

through an attack of cholera. He added that he had Bright's Disease, and he apparently had it. Assuredly he never had any diuretic stronger than milk from me.

When the kidneys first begin to act, we have always to watch the condition of the bladder, and, when needful, to relieve it by catheter. Otherwise, it is likely to be unable to act and to become greatly over-distended.

As regards the liver, there has always been a tendency to use calomel in cholera. When I arrived in India in 1848, I gave it as others did, in frequent large doses, ten grains to a scruple, to *compel* the liver to act. A few years later, Ayres's plan of giving calomel in frequent small doses, with the view of *soliciting* hepatic action, came into vogue. Subsequently, I preferred, when the bilious stools were either absent or small, or scanty bilious vomiting took their place, to use free counter-irritation by sinapisms, followed by large hot poultices over the whole hepatic region, front and back, and to solicit hepatic action by frequent gentle aperient doses and enemata.

Dr. Aitken says that, in men of intemperate habits, we often see, during the stage of reaction, obstinate vomiting of thick, tenacious, green paint-looking matter, probably bile-pigment acted on by some acid in the stomach or alimentary canal. This is, he considers, a symptom of evil omen, and it often goes on uncontrolled until the patient dies exhausted, and this, although all other symptoms may promise a favourable issue. It may last for a week, resisting all remedies, and proving fatal when the urinary secretion has been restored and all cerebral symptoms have subsided. In England, where cirrhosis of the liver and Bright's disease are much more common than they are in India, this irrepressible vomiting, in men of intemperate habits, probably depends mainly upon the presence of those diseases; as we find in India that where, in strong-looking dysenteric patients, the stomach persistently resists the use of ipecacuanha, the liver is nearly always considerably diseased. My Indian experience gives me no such terror of obstinate *bilious* vomiting after cholera. Indeed, I am usually glad to see bilious vomiting, regarding it as a very useful flux, only a little in the wrong direction. Here, under the free employment of sinapisms to the hepatic region and pit of the stomach, the evidences of gastric irritation generally subside, and a few salt-and-water enemata effectually solicit the bile to take its natural downward course.

As it is of great importance that we should view this *bilious* vomiting in its true light, I will quote what Dr. Goodeve says of vomiting in the reaction stage: 'Irritability of the stomach may be caused by some degree of congestion of its mucous membrane approaching to subacute gastritis, owing to the frequent straining and vomiting, and to stimulants incautiously given. It is not necessarily accompanied by feverishness, but there is generally thirst and burning heat of œsophagus

and at epigastrium. The patient cannot retain nourishment at first, the smallest amount being at once rejected. This condition often lasts several days, and requires great care and attention. I do not remember to have seen it fatal. When existing as the only symptom, great debility attends it, and convalescence is often delayed many days by its continuance. Sometimes it passes into a dangerous state of gastro-enteritis.'

This description applies to vomiting which has its origin in congestion and irritability of the stomach itself, and not to that vomiting which is caused by a copious regurgitant flow of bile. Still, it fully confirms what I have observed, that it is generally within our power to conduct the vomiting which occurs in the reaction stage of Indian cholera to a successful issue.

My own experience of the state of the bowels in cholo-uræmia is that their action is generally deficient: either there has been constipation, or the stools have been few or scanty.

One of our greatest difficulties in treating cholera arises from the fact that, in Bengal at least, the type of the disease changes, as I have already shown, from year to year, and even from week to week; nay, carefully observed, no two cases of cholera are precisely the same. Familiar as I was with the cholera of Calcutta, the disease which I treated during a great part of 1874 was of a type altogether new to me. It was, doubtless, owing to this variation in type that Dr. Goodeve wrote: 'The bowels' [in cholera uræmia] 'are sometimes relaxed, sometimes constipated; the evacuations yellow and feculent; diarrhœa may carry off some urea, and should not be checked.' He does not appear to have used purgatives in the cholo-uræmia of cholera.'

As then it is generally found that, when cholo-uræmia sets in, the bowels are more or less confined, the prompt use of castor oil in very moderate doses (two or three drachms), and warm enemata of common salt and *conjee* (rice) water relieve the head symptoms and produce five or six more or less bilious stools in the twenty-four hours. It cannot be too emphatically insisted on, that *prolonged constipation, in the reaction stage, is always a cause for anxiety*. Restoration of the excretion of bile is quite as important as the renewal of the excretion of urine. No fear whatever of relapse, or even of moderate gastric or intestinal irritation, need attend this evacuant treatment, unless we overdo it. In using moderate enemata, we are to bear in mind that the lower bowel has had little or no concern in the recent cholera flux. Hence it may be gently stimulated into action, with a view to a reflex impression upon the liver, without the slightest danger.

Indeed, I believe that, in cases where such gentle evacuant measures fail, stronger purgation is indicated.

We have reason to believe that there is no hope of recovery from cholera in cases where there has previously existed grave organic

disease of the kidneys and liver. It is probable that patients with confirmed morbus Brightii never recover from cholera.

In insisting that an evacuant system is needful in the cholo-uræmia of cholera, I in no way subscribe to the principle of the evacuant system of Johnson, as I have recourse to it not with a view to driving out the specific poison of cholera, but for the purpose of disembarassing and assisting the system in its efforts, as recovery advances, to free itself from accumulated and retained urine and bile-elements.

The utmost harm is done in cholera by the use of astringents, narcotics, and stimulants *after reaction has set in*, and by irrational attempts to stop vomiting and purging in and after the reaction stage. It is not for us to check nature's own means of clearing the system.

The clinical experience of several cholera seasons gradually established and confirmed in my mind the conviction that we can only prevent the cholo-uræmia of cholera, and successfully treat it, when we find it to be present, by a full recognition of the principles set forth above, and by watching the fluctuating conditions of the alvine and urinary excreta as narrowly as seamen watch the glass in threatening weather.

The vast importance of a course of treatment which will enable us to control the cholo-uræmia of cholera is shown by Dr. Goodeve's statistics of deaths during the reaction stage. In the great home epidemic of 1853-56, 14 per cent. of the deaths were from consecutive fever. In that of the North-West Provinces of India, in 1861, 22 per cent. of the fatal cases died in the reaction stage. Dr. John Macpherson shows that, during a period of ten years, one-fifth of the fatal cases of cholera in the Calcutta General Hospital died after the stage of collapse was over.

It is always important to bear in mind that, in cholera, as in true enteric fever, the main incidence of the disease is invariably upon the lower part of the small intestine. Here, principally, the rice-water stools are formed; and, as I mentioned above, this part of the bowel was absolutely occluded in the fatal cases which I examined post-mortem in the Bengal epidemic of 1849. Consequently, we should always examine the right iliac fossa, and counter-irritate there most freely, especially if there be fullness and tenderness.

Although I regard cholera as a pernicious fever, I have seen and heard nothing to show that quinine has any power of commanding it. Should cholera arise amongst us, every member of our community ought to take a tonic dose of quinine daily. Thus the lives of multitudes would probably be saved, the drug acting as a nervine tonic, promoting healthy digestion. But, as we have already seen that quinine is not *specifically* prophylactic in averting marsh fever, it cannot be relied upon alone as a certain means of preventing cholera (Macpherson).

We, of course, give quinine as a tonic in convalescence. In the

outbreak of pernicious fever at Deesa in 1835, the symptoms of which have been detailed above (pages 184 and 280), quinine was pretty freely used, but 'often disappointed expectations.' But, at that time, quinine was not relied upon as it deserved to be. In describing the fever at Bellary, in 1840 (page 279), Mr. Parry, although a strong believer in calomel, places the following words in italics: '*During the intermission it*' [calomel] '*was frequently combined with quinine, and in this combination it proved eminently serviceable.*' Speaking of the 'Malwa Sweating Sickness,' Dr. Murray wrote: 'Quinine is the sheet-anchor in this disease, and there are few symptoms that would prevent my giving it.' Hence the importance of judging, in any outbreak and in every case, whether we have to deal with pernicious fever or true cholera. 'Cholera,' says Macpherson, 'is not ushered in by rigors. The fluids ejected are yellow, green, or bilious in pernicious fever; in cholera, like rice-water.'

All my experience tended to show that few things avail more in the management of cholera than *sedulous care and good nursing*. I often told my students that, if I should be attacked, I should wish to be attended by one who imagined that he had discovered a cure for the disease—no matter what, unless it happened to be croton or castor oil, nitrate of silver, or tincture of lytta.¹ The discoverer, when he is, as he usually is, a man of experience, science, and humanity, always surrounds his 'cure' with so much wise precaution and sedulous care, and such attention to every turn of the malady, that his patients undoubtedly stand a better chance of recovery than do those who come under a routine system of everyday treatment. Whatever his plan of treatment may be, he will, assuredly, have the largest amount of success, even when the outbreak is at the deadliest acme of its destructiveness, who treats every case as a distinct therapeutic study. This law is enforced by the fact, which I have already repeatedly insisted upon, that no two types of cholera—nay, that no two cases of this disease are precisely the same. Hence the necessity of not relying wholly upon any specific or routine mode of treatment, but of treating every case throughout in strict accordance with its own particular conditions. On the other hand, Indian physicians are so fully accustomed to the disappointment of losing nearly every case at the first outbreak of cholera, that this ill-success never shakes their confidence in what they have proved to be right principles of treatment. If we treat our cases steadily upon a system proved by long experience to be rational, we shall probably find that, at the end of an outbreak, at least some 40 per cent. of our cases have recovered. If we change our plan indecisively every time we lose a case, we shall, at length, have the conviction forced upon us that our treatment has failed throughout, and that we have left a vast amount of good undone in taking the responsibility which ought to have been held by a man of decision.

¹ All of which enjoyed brief reputation.

I know of no disease in which so much benefit may be obtained from *good nursing* as in cholera. Sensible nurses may be readily trained, and need have no apprehension whatever that this duty will, in the very slightest degree, add to the danger which they share with every other member of the community. In giving ice, in seeing that the patient does not incur the peril of sudden death by syncope on having his head raised, in attending to the heat of the water-bottles and in applying the sinapisms, in keeping to their work the relays of assistants who apply the dry ginger frictions, in feeding, in encouraging the patient, and indeed in carrying into effect every means of relief and every needful precaution until convalescence is established, an active and experienced nurse is invaluable. It was my frequent remark in India that my leading measure in the care of my cholera patients was the attendance of Mrs. Sheen, the practised nurse of my cholera ward.

I must repeat that, for a considerable time before I left India, I considered that I possessed certain definite indications which guided me in the treatment of cholera.

I will not boast that, from the time at which I first entertained these views, and carried them unswervingly into practice, I achieved any marvellous success in the treatment of the cholo-uræmic stage of cholera. I lost a very few patients from cholo-uræmia. I, however, felt that my failures were almost invariably due either to the fact that the patients had faulty organs, or to the circumstance of their having been brought to hospital too late for relief. Whenever these views had fair play, they led to decided success. I ceased to regard this cholo-uræmia as a terrible and irresistible mystery. I felt that I could generally either prevent or encounter it successfully in a patient of fairly sound constitution, whom I treated from the commencement of the attack.

Nourishment in Cholera.—It is unquestionable that many deaths from cholera, whether they occur in the stage of collapse or in that of reaction and cholo-uræmia, are, potentially, in a large measure due to starvation. The disease, in its early evacuant violence and paralysis of absorbent action, drains the blood of its fluid, and deprives the solids of the body of all nutriment. If, in the stage of collapse, we give milk, soups, more iced water than a table-spoonful at a time, &c., by the mouth or rectum, they are, in my experience, immediately rejected; and, with each vomit and dejection thus excited, the patient's strength goes down—as if, instead of endeavouring to feed him, we bled him. As long as the kidneys and liver are absolutely unable to act, we can scarcely hope to do much towards nourishing and supporting the patient by ordinary 'feeding.' We, of course, begin cautiously to give nourishment (I preferred milk), in small quantities frequently, as soon as we find that it can be retained; but effectual means of nourishing in cholera are still an unattained desideratum in thera-

peutics. At present, our main consolation is that we have seen hundreds of patients 'pull through' attacks of algide cholera, in which it has been evident that we were unable to effect much in the way of nourishment until the liver and kidneys had begun to resume their functions. With a heroism which has never been wanting among Indian surgeons, Dr. David Boyes Smith, of Netley, while acting for me, some twelve years ago, as Senior Physician of the Medical College Hospital at Calcutta, gave his blood, I believe, to deliquium, which was transfused into the veins of a hospital patient in the collapse stage. I understood that this noble act injured the physician's health without materially benefiting the patient. I was told this by our students—never by Dr. Smith. It is not exactly blood that is wanting in the vascular system of the dying cholera patient, who retains his due quantity of fibrin and blood-discs. This is proved by the fact that, in Indian cholera, many recoveries take place after hours of algide collapse, and that, in these cases, discharges of hæmoglobin do not attend convalescence; hence we may consider that whatever injury the blood-discs may sustain during the collapse stage is reparable up to a very late period. In the present day, it does not occur in the experience of everyone to see the living blood of a collapsed cholera patient. Some years ago, it was suggested that stimulants should be given during collapse, and that, then, a vein should be opened. By this means, it was conjectured, the circulation would be freed and the heart's working power restored. I, acting carefully upon this suggestion, gave hot brandy-and-water, and opened a vein in the arm of a fine young English sailor in the collapse stage, his head being kept low. The blood was of very good colour—not 'tarry'—but it was distinctly thick, trickling down the arm, and ceased to flow before two ounces had escaped. I wish that it could have been fully examined; but, in our concern for the patient, who sank rapidly, we failed to preserve it. It appeared clear that this blood only wanted serum. I believe that, in cholera, the blood-discs live quite as long as the man does, and could receive and benefit by a supply of serum up to the moment of the article. What appears to be wanting here is a fluid capable of supplying, in every constituent, the place of that which has been drained away. But the composition of such a fluid and the means by which it may be introduced into the bloodvessels have still to be demonstrated. The practice of transfusing variously-composed saline fluids had been abandoned, as worse than useless, before I went to India. Milk transfusion has appeared to afford some very doubtful promise; but I think that everyone who studies Dr. Benjamin Ward Richardson's most suggestive commentary upon the whole of this question,¹ and reads Dr. T. M.

¹ *M. T.*, p. 124 *et seq.* of vol. ii. for 1883: 'On Feeding by the Veins, and on Intraperitoneal Injection in the Collapse of Cholera.'

Lownd's practical observations on 'Feeding Patients in Cholera Collapse,'¹ will consider that the renewal of the lost constituents of the blood and the administration of direct nourishment during the stage of collapse are points largely open to hopeful research. I believe that few could do more justice to such an inquiry than the advocate of peptonised food prepared by the pancreatic method²—Dr. William Roberts, of Manchester.

In closing this chapter, I must repeat that no system of treating cholera can be either scientific or valid unless every step be taken with due regard to the conditions of the kidneys and liver.

The 'Comma' Bacillus in Cholera.—In the summer of 1885, I was a member of a committee convened by the Secretary of State for India for the purpose of taking into consideration a report by Drs. E. Klein and Heneage Gibbes, entitled 'An Inquiry into the Etiology of Cholera,' sent in by those authorities upon the conclusion of their work of investigating this subject in India.

Our committee were quite unanimous in arriving at the following conclusions :—

'The investigations described, like most others recently undertaken with a view of elucidating the etiology of cholera, may, for the most part, be characterised as an attempt to confirm or refute the doctrine that the disease is caused by a microscopic comma-shaped organism. The results of these and of others of allied nature which have been brought to the special attention of the committee may, briefly stated, be summed up as follows :—

'1. That comma-shaped organisms are ordinarily present in the dejections of persons suffering from cholera ;

'2. That they are not to be found in the blood nor in any of the tissues, including the mucosa of the small intestine when the latter is examined in a fresh condition ;

'3. That comma-shaped organisms of closely allied morphological appearances are ordinarily present in different parts of the alimentary tract in health ; that they are developed to an unusual extent in some of the diseases characterised by hyper-secretion of the intestine ; and that there are grounds for assuming that when any predominant form is observed it is in great measure attributable to the nature of such secretion ; and

'4. That the comma-shaped bacilli ordinarily found in cholera do not induce that disease in the lower animals, and that there are no real grounds for assuming that they do so in man, whilst the circumstance that they have been found in tanks which constituted the ordinary water-supply of adjacent villages unassociated with the presence of the disease, goes to negative any such assumption.'

¹ *Lancet*, p. 123, vol. ii. of 1883.

² *Transactions of the International Congress of 1881*, vol. i. p. 517.

‘Drs. Klein and Gibbes have made a valuable contribution to our knowledge of the bacterial organisms associated with cholera, though the evidence hitherto adduced does not warrant the conclusion that any of them bear a causative relation to the disease. As regards the question of its essential cause, the committee are glad to learn that the Government of India are making further arrangements for having investigations, of a varied character, continuously conducted in that country under the direction of Dr. Douglas Cunningham.’

Each member having been invited to express his own views on the subject, I submitted the following note :—

In considering this report, the following leading facts claim our attention.

It has, for many years before the appearance of Dr. Koch’s observations on the subject, been a matter of demonstration that various microbes occur in cholera stools.

In Egypt and Calcutta, and subsequently in Toulon, Dr. Koch found that ‘the comma-bacilli are constant concomitants of the cholera process.’ The researches of Drs. Klein and Gibbes, conducted in Calcutta and Bombay, have confirmed this observation.

These three observers are now agreed in stating that ‘comma-shaped bacilli occur also in other conditions.’

Bacilli, very similar to these, occur in the human mouth. Others of like appearance have been found in tank water.

These micro-organisms have not been found in the blood of cholera patients.

Their introduction into the systems of lower animals has not produced cholera.

The few data which we possess with regard to the effects produced by the introduction of these microbes into the human system are fraught with uncertainty.

Having accepted these facts, it appears that our committee have, in considering the statement of these two sets of observers, no perfectly solid ground to advance upon. It is evident that more facts are needed, and that, until these are established, opinions formed upon these data would be premature.

The report under consideration has validly advanced that course of research which Dr. Koch’s statements demand from pathologists ; but it is to be regarded as being essentially a paper of progress, as one of the earliest and probably most suggestive of the series of observations which this subject still demands.

Allowing to each its full value, the inquiries of Dr. Koch and of Drs. Klein and Gibbes must be regarded as, unavoidably, wanting in strength and conclusiveness, because the field of each set of observations was limited in respect to locality, season, and type of disease.

Cholera varies in its type more than any other disease with which we are acquainted. I watched cholera in its 'endemic area,' Lower Bengal, during twenty-five cholera seasons, and I never saw precisely the same type of disease or exactly the same morbid changes in any two outbreaks or in any two cases. My large experience of the coarse morbid anatomy of cholera would not enable me to predict, with anything like an approach to accuracy, the intestinal lesions which would be most prevalent in any coming outbreak.

Hence, doubtless, the want of agreement, as set forth at pages 7 and 8 of the report, between the two sets of observers, in regard to the morbid conditions in which they found the ileum.

Consequently, to be conclusive, practical inquiries into the etiology of this disease must be conducted in many distinct localities, in many outbreaks, and by many observers. Probably the Indian Government will consider such a line of inquiry needful. The validity of Dr. Koch's observation is not shaken by the reporters' statement, at page 7, that micro-organisms, undistinguishable from the comma-bacillus of cholera stools, occurred in an autumnal epidemic of diarrhœa in Cornwall in 1883, and in several cases of dysentery, because our knowledge does not enable us, in all cases, to distinguish epidemic diarrhœa from cholera, and because the experience of every Indian observer teaches him that the subjects of chronic dysentery are very liable to fatal attacks of cholera.

The facts adduced by Inspector-General Lawson at the last meeting of the Epidemiological Society (*Trans.* for 1884-85, p. 102) negative the reporters' statement, at page 4, that 'notorious immunity from cholera' is enjoyed by ships on the high seas.

Upon the question, Does the acid of the gastric juice kill comma-bacilli? I may refer to a fact which I stated two years ago¹ viz., that we have evidence that, at the outset of cholera, digestive power appears to be wholly in abeyance, so that the material of the first vomit may consist mainly of an immense meal of meat and vegetables returned without the slightest appearance of digestive change. Interesting as it is, the observation that the comma-bacilli invariably occur in Asiatic cholera, assuredly does not prove that this micro-organism is the germ or contagium of cholera, or that, when introduced into the human body, it is capable of producing cholera, acting as a specific poison. To maintain this position, its supporters must introduce the cholera bacillus into the stomachs, say of criminals, under various conditions of health, of fasting, and of digestion. These experiments should always be made, first, during a cholera outbreak, and then in localities which are, at the time, free from cholera.

While we may view with interest the curious rather than important fact observed by Dr. Koch and confirmed by the researches of Drs.

¹ *M. T.*, vol. ii. for 1882, p. 342, and pp. 293-94 of this volume.

Klein and Gibbes, we are not, at the present stage of the inquiry, warranted in concluding with Dr. Koch that cholera is contagious, and that the cholera bacillus is probably the germ or contagium of cholera. I, for fifteen years, held charge of a cholera ward in the largest hospital in India; and, having the question, Is cholera communicable from man to man? constantly present in my mind, I am confidently of opinion that there does not exist, in the body of a cholera patient, any poison which, introduced into the system of another individual, will act as the specific poison of cholera.

To non-medical persons, the fact that a certain microbe is always found in cholera is not unlikely to lead to the very common unscientific error of confusing coincidence with cause. Thus it has long been noticed in India that the larvæ of the blow-fly not unfrequently fill the nasal passages. Here, it might be said, 'flies enter the nose in sleep and cause the disease *peenash*; consequently, everyone ought to plug his nostrils on going to bed.' Investigation of these cases has, however, proved that healthy persons are not liable to this condition, which only occurs in the subjects of *ozæna*, arising from disease of the nasal bones attended with a putrid discharge which attracts the flies.

Living, as I and my family did for many years, with absolute impunity, in quarters close to a hospital which, during that term, contained many hundred cases of Asiatic cholera, I, of course, absolutely disbelieve in sanitary cordons and in systems of quarantine as means of defending countries from cholera invasion.

Localities exempt from cholera visitation.—In a pamphlet published in 1852, Surgeon-General Edward Balfour propounded the question: *Are there towns or villages in India where cholera has never appeared from the period of its first outbreak in 1817?* Dr. Balfour published in 1856 an extension of this inquiry: 'The Localities in India exempt from Cholera.' I gave so full an analysis of these invaluable observations in my work on 'The Means of Preserving the Health of European Soldiers in India'¹ that I need not do more here than draw attention to such facts adduced therein as that the Cantonment of Palaveram was reported to have escaped the attacks of cholera for the preceding twenty-five years; and that the mountain range in Orissa, inhabited by the Khonds, had never been visited. The interest and importance of Dr. Balfour's most extensive and laborious researches into this great question are much enhanced by the fact that, in reply to my inquiry as to whether subsequent experience has confirmed his results, the Surgeon-General has written to me (February 18, 1886): 'I think that my inquiries as to places in India exempt from cholera were carried out with every precaution needed to secure correct information; and, in all the years that have passed since I wrote on the

¹ *I. A.*, No. 11, p. 271.

subject, I have not observed that the facts which were then made known have been questioned.'

As it has, I believe, been stated that certain large English towns, especially Cheltenham and Birmingham, have been exempt until now from visitation by cholera epidemics, I have lately made inquiries of medical friends long resident there. In reply I am informed by Dr. John Bassett, of *Birmingham*, that cholera has never been epidemic there. He cannot learn anything about the visitation of 1832; but in 1849, some half-dozen cases happened in the old workhouse, and solitary cases were met with in the borough of people who had come from cholera districts. In 1866, some isolated cases were investigated by the coroner. They were all imported cases. Dr. Bassett adds that he has discussed the cause of this immunity with Dr. Hill, the officer of health, who ascribes it to the high level on which the town stands. Alluding to the law that cholera follows the course of great rivers, Dr. Bassett says: 'We have only a brook here; but then the same may be said of Bilston, a few miles hence, and there the cholera has been virulent on such occasions. Dr. Hill explains this by showing that Bilston is on a lower level and is flat.'

Of *Cheltenham*, Dr. John Craig, a medical officer of great Indian experience, tells me: 'You may depend on the statement that epidemic cholera has never visited Cheltenham.' 'In the last visitation of the disease, there were cases in some villages of Gloucestershire, as Berkeley and Wotton-under-Edge, on this' [*Cheltenham*] 'side of the Severn; whereas, on the other, and over the whole county of Hereford, there was not a single case. I was told by a proprietor in that county that the bishop had a form of prayer of thanksgiving on this account.'

If the attention and practical action of persons in authority could be drawn to my recommendation of establishing medico-topographical and health histories for districts and towns, published in the 'Transactions of the Epidemiological Society for 1883-84,' the profession and the public generally might learn that many other localities besides Birmingham and Cheltenham and those cited from Hirsch and Cunningham in Sir J. Fayrer's recently published lecture on 'The Origin, Habits, and Diffusion of Cholera,' page 22, are to be viewed as 'cities of refuge' during visitations of this pestilence. See note, p. 176 of this volume.

ERYSIPELAS.

In 1854, Kenneth Mackinnon remarked:¹ 'The idiopathic erysipelas as it appears on the face and lower extremities, unconnected with wounds, is a rare affection in India, and I know of no instance of its epidemic appearance. Erysipelas attacking wounds I have fre-

¹ *I. A.*, No. 3, p. 177.

quently seen prevail as an epidemic, exciting in the merest abrasion of the skin, as in the sores on the legs of prisoners from the chafing of their irons, high and diffuse inflammation, requiring free incisions to prevent sloughing. It has been during humid sultry weather, and especially at the breaking up of the rains, I have seen wounds take on this action in an epidemic form. In the Medical College Hospital and in the Native Hospital in the Durrumtollah, wounds made in operating are apt to take on this action, especially in the rains.'

Morehead (p. 200) agreed with Mackinnon in considering that idiopathic erysipelas was, in his time, rare in India. He met with very few cases in Bombay, either in Europeans or in natives. He added that traumatic erysipelas is of more common occurrence, and at times evinces almost an epidemic tendency. It was common in the Jamsetjee Jejeebhoy Hospital in November and December 1851, after wounds of the scalp and lower extremities, but was easily subdued. It did not in all cases originate in the hospital, but, in some, was present on the admission of the patient; thus showing that it was not, at least invariably, due to the air of the hospital. On one or two occasions, he also noticed the liability to erysipelas after application of blisters, so marked as to render it expedient to discontinue their use for the time.

In my considerable Indian experience, between 1848 and 1873, so little *idiopathic* erysipelas appeared among my own patients, that I used to speak of a solitary (facial) case which had occurred in my medical wards in the Calcutta Medical College Hospital, which has 300 beds. In the eight years 1865-72, however, sixteen *traumatic* cases proved fatal in the surgical wards. In 1873, a wave of erysipelas arose in Calcutta; and, from February 1874, this disease appeared to have taken up its abode in the surgical wards of the hospital, spreading from case to case, and attacking traumatic cases so frequently that the surgeons became apprehensive that it would occur whenever they were obliged to operate. I then foresaw the appearance in our obstetric wards of puerperal fever. In the first week of June, that disease became so prevalent there that Dr. Charles was obliged to remove his lying-in women to a separate building.

There was no accession of idiopathic cases to my medical wards.¹

In his annual report of the General Hospital, Madras, for 1882, Surgeon-Major R. W. Cockerill observes that 'regarding erysipelas, the experience of the past year agrees with that of all former ones. This disease is a rare one in Madras, and never attacks the patients in hospital. All the cases (four in number, all Europeans) came from outside; and, although the patients are generally treated in the ordinary wards, such a thing as the disease being conveyed from one patient to another, by infection or contagion, is unknown.'

¹ For a detailed report on the outbreak, which I made to the head of the medical department, see *I. M. G.* for 1875, p. 314.

The returns for 1881 show that 8 *European Soldiers* died of erysipelas, the distribution of cases being : Bengal, 2 ; S. Afghanistan, 1 ; Madras, 3 ; Bombay, 2. Of *European Women*, 8 were admitted in Bengal, of whom 1 died, and 2 were admitted in the Bombay Presidency. Total, 10 cases, and 1 death. Of *Soldiers' Children*, 5 were admitted and one died ; 3 admissions with 1 fatal case in Bengal ; 2 admissions in Madras. Of *Native Troops*, there were 51 admissions in Bengal and 4 deaths. In the Punjab Frontier Force, 38 admissions, and 3 deaths. Hyderabad Contingent, 1 admission ; Bombay, 7 admissions ; Madras, 9 admissions, 3 deaths. In the *Gaol Population*, there were 150 admissions in Bengal, 29 deaths ; 12 admissions, and 2 deaths in Bombay ; in Madras, 6 admissions, 1 death ; in British Burmah, 2 admissions ; in the Andamans and Nicobars, 7 admissions and 1 death.

RHEUMATISM AND GOUT.

Rheumatismus Acutus, see Diseases of the Heart.

Writing in 1835,¹ Mr. Malcolmson, of Madras, said that the common acute rheumatism of Europe is very little known in India. In reviewing this work, Dr. Corbyn, whose Bengal experience was large, by no means opposed this view. Morehead, writing from experience in Bombay, says : ' It may be that *acute* articular rheumatism is not so common in India as in colder climates, yet it is by no means rare ' (p. 561). I saw it very distinctly, with its cardiac complications only too well marked, in Calcutta, but only in three or four (cardiac) cases. In a European, there was the characteristic sweating, which I think I could not mistake, but the attack passed off in two or three days.

Reporting in 1839, Dr. John Murray, senior, stated that the ' Service in Madras were losing more in efficient strength by rheumatism than by any other class of disease.'² He attributed this mainly to the unfitting state of the barracks and guard-rooms in that command. Although great improvement has been effected in military buildings, the prevalence of rheumatic disease is still great. In the year 1881, the admissions for rheumatism among the European troops in all India numbered 2,757, or 47·0 per 1,000 of strength. No distinction of acute and chronic is made in the tables ; but the fact that *there were no deaths* appears to show that true rheumatic fever was neither prevalent nor grave in type. The invalids under this heading amounted

¹ *Observations on Some Forms of Rheumatism Prevailing in India.*

² Reporting from Bellary (Madras Presidency), Dr. Allman said that rheumatism was the most troublesome and tedious disease which the army medical officer has to deal with in India. Although in very few instances fatal, it deprived the army annually of many soldiers.

to 1.48 per 1,000 of strength. The admissions of European women were 40 in 3,741, with no deaths; of children 10 in 6,548, no deaths. Among 100,844 prisoners, there were 447 admissions for acute rheumatism and only one death. The admissions for chronic and muscular rheumatism were 832 and 564 respectively. The incidence of rheumatism upon the prisoners in the Andaman and Nicobar Islands, averaging 11,225 strong, was remarkably heavy. The admissions were: acute, 31; chronic, 424; muscular, 207; total, 662, or 65.5 per 1,000; but no deaths.

Synovial Rheumatism of the smaller joints, as those of the phalanges of the fingers and toes, as well as of the larger synovial sacs, as the shoulders and knees, is frequent among Europeans who have long resided in the damp, malarious, and inconstant climate of Bengal. Although these painful affections are often very chronic, laming people, and resisting all treatment for years, they, like muscular rheumatism, seldom, if ever, cause material lesions in affected joints, and at length disappear, generally suddenly, of their own accord. I know persons, long retired from India, who, having suffered in almost every joint, are now free from pain or stiffness in any articulation, but who are liable to be attacked at any time, especially in damp and cold weather, with pain and disability almost anywhere, but most particularly in those joints which have hitherto suffered least.

Muscular rheumatism is of very frequent occurrence during the rainy season in Lower Bengal. Under the head of Tetanus, I have spoken of a singularly marked coincidence, indicative, as I believe, of etiological affinity, which is observable in the appearance of these two diseases. I think that there are now few large muscles in my own body which have not been subject to this malady, which I have endured without any attempt at medication, in the certainty that nature would relieve me. In my last voyage from India, the heat was so great that I could not occupy my berth, and lay in an exposed position on the deck under an open hatchway. The result was a pain in my left popliteus muscle which troubled and considerably lamed me for many months after my return. As I was beginning to think that I was crippled for life, still refraining from treatment, the affection suddenly disappeared and has never returned in that locality.

I have noted a remark made by Mr. Morgan in a report on the state of health of troops at Cannanore,¹ which, although true and useful, must be accepted with some reserve:—

‘A hundred and forty cases of this class of disease have been treated, and two of them were invalided; no deaths. That most convenient complaint, “pains all over me,” in the mouths of soldiers who think they have completed service long enough to entitle them to discharge, has and will cram the wards of a hospital in a station

¹ *I. M. J.*, vol. v. p. 335, N. S.

where the facilities of "catching cold" are certainly great during the rainy season. Such cases are really quite puzzling. There is no violence of inflammatory action to control; the secretions are all healthy in quantity and quality; yet the "pains" go flying about from head to foot in all directions. If we blister or leech the loins, the most common site, in ten days we have the shoulders implicated, and *vice versâ*.' Mr. Morgan evidently regarded most of those who thus complained as malingerers, and probably a large proportion of them were; but such is the natural course of the affection in genuine cases.

Gout.—Writing in 1837, Dr. Corbyn remarked that this is a disease scarcely known among the inhabitants of warm climates. He added that East Indians are rarely affected with gout. A young Englishman, born of gouty parents but still free from joint disease, may enter upon a career in Lower Bengal with a strong hope, that, with prudence, he may escape the threatened evil. I believe that gout scarcely exists among the natives of India (Dr. Irvine wrote, in his 'Medical Topography of Ajmeer,' 'I have heard of gout occurring among some of the Rajpoots who drink hard, but I never met with any such cases'), and, although I have seen one or two very moderate cases of this disease among Europeans, and have heard of a very few others, I think that, with all its sanitary faults, Calcutta is an earthly paradise for those cautiously-living people whom gout threatens, but has not crippled. Before I went to India, I saw very little of the diseases of the rich. In the East, I saw much of the ailments of ladies and gentlemen born and living in wealth, but it was not until twelve years ago, in helping an old gentleman with unmistakably characteristic hands and feet into a Bayswater omnibus, that I said to myself, 'I now see typical gout for the first time!'

I have already mentioned that men who have escaped gout in India are very liable to it when retired in England. Just as I am writing this, I have chanced to meet, in a single week, a colonel and two major-generals who are thus circumstanced. A young English millionaire, on finding that he is threatened with a life of suffering from gout, could not do better than take up his abode at Calcutta.

It is only right to add that Surgeon-General Moore has recently stated¹ that he believes 'gout will be developed sooner, and in a more irregular manner, in those predisposed, if residing in a tropical climate.' I do not think that this doubtless just observation is at all applicable to the sub-tropical climate of Calcutta.

I have from time to time watched the sufferings from *Gouty Synovitis* of an esteemed brother officer. About nineteen years ago, Dr. —, a remarkably well built and agile man, was caught when walking on the Midaun (plain) in one of the most terrible cyclones that has visited

¹ 'The Constitutional Requirements for Tropical Climates,' *Trans. Epidem. Soc.*, vol. iv., N. S., p. 41.

Calcutta in modern times. He stood long, clinging to a lamp-post, exposed to the fury of wind and rain. The consequence was an attack of acute synovitis of a knee-joint. In subsequent years, other joints have become affected; but he recovered well from repeated attacks; and, when I last saw him, it was clear that no large amount of deposit had occurred. These attacks are undoubtedly gouty.

SYPHILIS.

Those whose experience has been principally obtained in large Indian stations, or who, like myself, have practised chiefly in much frequented Oriental ports, have not, I believe, observed any remarkable differences in the type of the primary and secondary forms of this disease as they occur in Europe and in the East. Still, as our acquaintance with the remote hill tribes of India is daily upon the increase, it would be well if, while a community is still in its primitive condition, inquiry could be made into the prevalence of syphilis among them and into the characters of the disease as it now exists.¹

Formerly, it became a matter of observation by surgeons, especially in the Madras Presidency, that syphilis has greatly increased in prevalence and become aggravated in type during famine seasons. Dr. John Clark, of H.M.'s 13th Light Dragoons,² showed that, while his regiment was stationed at Bangalore, during the dreadful famine of 1833, the poor and half-starved village women were known to cohabit with the soldiers for a handful of grain. He adds that prostitution prevails to a great extent at such periods, and the state of debility induced appears to be favourable to discharges from the sexual organs of the female. He gives a return of admissions for syphilis and gonorrhœa in the regiment, showing that—whilst, during ten of the years between 1820 and 1834, the number of cases ranged from 63 to 110 annually, only in one year reaching 140—the admissions during the four exceptional years, *which were years of famine*, ranged from 226 up to 350.

In this report, Dr. Clark mentions a fact which is still noteworthy. From observations made in the 7th and 81st Regiments, he found that 'more cases (of syphilis) often occur in one regiment in the East Indies than in the whole of the West India command.' Dr. Clark assigned this difference to the infrequency of prostitution among the negro slave population of the West, 'there being little or no encourage-

¹ For a series of valuable observations on the characters of syphilitic sores, as they appeared during a series of years in regiments on home and Indian service *vide I. A.*, No. 5, p. 20:—General statistics of 'Local Venereal Ulcers,' 'Syphilitic Bubo,' and 'Consecutive or Secondary Syphilis,' as met with among soldiers in the United Kingdom and in India, by C. A. Gordon, M.D.

² *Madras Quarterly Medical Journal* for October 1839.

ment for prostitution amongst a people who are clothed, fed, and encouraged to marry.' The number of cases, for all regiments in the West Indies, in 1823, was 36 ; the returns of the 13th Light Dragoons in India for the same year showed that there were 44 cases of primary syphilis *treated*. The average of syphilitic cases in that regiment during eighteen years, 1820 to 1837, was 61, the average strength of the regiment probably not being above 800.

A report by Mr. McGregor on Syphilis in H.M.'s 39th Regiment from 1833 to 1838, during the greater portion of which term they were in cantonments at Bangalore,¹ gives the very high average of 191 per mille for the six years. These averages may be usefully compared with those of the army of India of the present day, viz., primary, 87·6, secondary, 23 per mille.

All observation combines to show that syphilis rises to its greatest prevalence and malignity in armies worn out in long campaigns, and whenever the men's constitutions are tainted with scurvy. I have cited elsewhere facts leading to this conclusion.²

In the year 1860, when the measure of re-establishing Military Lock Hospitals in India was under discussion, I collected with much care and published³ materials for a history of these establishments. Now that the Lock Hospitals in the United Kingdom and in India stand nearly useless, I would earnestly solicit the attention of the authorities and of army surgeons to that very striking array of facts. In 1797, the Governor-General established *hospitals for diseased women* at the stations of Dinapore, Berhampore, Cawnpore, and Futtighur. Some thirty years subsequent to their establishment, these institutions had fallen into disrepute, assuredly under maladministration. Accordingly, they were abolished, in the Bengal Presidency, in 1830, but were permitted to survive in the presidencies of Madras and Bombay until 1835. Many medical officers of great ability and experience—Dr. Murray, senior, Surgeon Sievwright, Dr. Clark, Dr. Mouat, senior, and Dr. Arnott—deeply regretted the abandonment of this system and showed that an increase of syphilis was the result. I have fully cited their statistics and arguments. I have also shown that, being virtually left under the control of a native doctor and a female assistant, both people of the lowest class and upon the very smallest allowances, these places became dens of iniquity, and their keepers agents of extortion and oppression. The only surprise appeared to be that they could possibly have been maintained for nearly forty years in every large military station throughout India. My late honoured chief, Sir John Forsyth, Director-General of the Medical Department, held that the whole history of Lock Hospitals in India distinctly proves that such estab-

¹ *I. M. J.*, vol. vii. p. 257.

² 'On the Means of Preserving the Health of European Soldiers in India,' *I. A.* No. 13, p. 230.

³ *Ibid.* p. 298.

ishments, if conducted upon the old system, must almost inevitably be failures, as, with the exception of the European medical officer in charge, the whole system was carried on by native agency. The system of placing the women of the Regimental Bazar under the supervision of the quartermaster, and under the inspection of one of the medical officers, and those of the Sudder Bazar under the surveillance of the cantonment magistrate, was found to keep the Bazars tolerably free from disease. This eminent authority held that the only prospect of making a Lock Hospital really useful is by conducting it with the vigilant and zealous co-operation of these two officers; and, even then, little good can be expected unless a medical superintendent of standing and activity is placed at the head of the establishment, assisted by a European matron; and, to be perfectly successful, even this scheme must have the hearty support of the whole of the regimental and medical authorities on the spot. The Director-General was 'fully impressed with the necessity of adopting some such decisive and comprehensive measure as that sketched above for the prevention of a disease which is known to undermine the health of so large a proportion of our European force.'

In the middle of 1859, a Lock Hospital at Meean Meer was sanctioned as an experimental measure. The system has been general until very recently.

Judged by the evidence of statistics, this much-debated experiment, pursued for nearly a quarter of a century, has not been successful. It is shown in the Eighteenth and Nineteenth Reports of the Sanitary Commissioner with the Government of India, 1881 and 1882, that, in the whole army of India during the six years 1876-81, the annual admissions per mille for *primary syphilis* have been at the rates of 60, 65, 95, 79, 88, 92, and 87·6. It has long been argued that, even if this system failed to diminish the number of cases, it would have the effect of rendering the type of the disease less virulent. As evidence that this improvement has not been effected, Dr. Cunningham shows that, during the above years, the ratios per mille of *secondary syphilis* have been 24, 22, 22, 24, 23, 23, 23. The admissions for gonorrhœa in 1880 were at the rate of 106·5, in 1881 of 107·3, and, in 1882, of 119·8, per 1,000. The Sanitary Commissioner observes: 'In noting these results, the facts only are dealt with, and they are most unfavourable. As pointed out in this office annual report for 1878, they may in some measure be accounted for by the increased youth of the army and by the smaller proportion of married men, but, making every allowance for these factors, *the failure of the Lock Hospital system is still most decided.*' [The italics are mine, N. C.]

In his report for 1882, the Commissioner writes: 'In the report for 1881, it was shown, from the statistics of venereal diseases, in the European army of India, during a series of years, that the Lock Hospital system

in the cantonments of this country has proved a failure. It was there noted that the admission-rate for these diseases in the army of India had been 260 per mille during the year 1881, and that, with the single exception of 1878, this ratio exceeded that of any year since 1871. Another year's experience confirms the conclusion then arrived at. During 1882, cases of venereal disease were more common than they had been even in 1881, and equalled 265 per 1,000. This admission rate . . . exceeds the average ratio of the ten-year period, 1870-1879, by 62 per mille.'

In the first draft of this chapter, published in August 1884, I remarked that it could hardly be doubted that, armed with these texts, those in power in the present day would take active measures for denouncing and crushing the Lock Hospital system in India, as their predecessors wrote and clamoured it down in the third decade of this century. I understand that one of the last acts of Lord Ripon was, in effect, to set these establishments aside.¹ As I was writing on this subject in 1884, I saw it announced,² of Lock Hospitals at home, that 'venereal of the most virulent type is still the prevailing disease among the troops at all the military stations.' 'The Lock Hospitals are almost empty. Attendance at these institutions being now optional, but few unfortunates present themselves for treatment, and these, as a rule, are only bad cases.' The writer adds: 'Fostered and encouraged as it is, this loathsome disease is spreading to such an extent as to cause grave anxiety.' A recent article in the '*Lancet*'³—after showing that the Select Committee of the House of Commons reported favourably of the Contagious Diseases Act, expressing an opinion that, by their means, venereal disease had been diminished, and that the efficiency of the army had been increased, on which grounds they declined to recommend their repeal—gives full statistical data in evidence of the facts that primary sores and secondary syphilis were much more frequent, in all the groups of stations, in 1882 than in 1870-72; the increase, at the protected stations, was 42 per cent. for the primary sores, and 31 per cent. for the secondary form of the disease, as against 60 per cent. and 22 per cent. for the fourteen unprotected stations; or, if all the unprotected stations be included, the increase for primary sores was 40 per cent., and for secondary disease 17. But it must be observed that, at both, the lower ratios of 1870-72 were much in excess of the higher at the fourteen protected stations in 1880-82. As

¹ The following rather singular entry occurs in the *I. M. G.* for January 1885: 'As the system of providing Lock Hospitals in connection with British troops has not been appreciated by the men, it has been decided, after a lengthened trial, to close these hospitals throughout Bengal.' In February 1886, the editor writes: 'The Calcutta Lock Hospitals are now entirely voluntary.'

² *M. T.* for January 26, 1884, p. 135, citing the *Army and Navy Gazette*.

³ For January 2, 1886, p. 23.

regards gonorrhœa, there was a decrease of 7 per cent. in 1880-82 at the fourteen protected stations, while, in all the other groups, there was an increase of 20 and 12 per cent. in the same period. It is, therefore, urged that, obviously, the operation of the Acts limited the prevalence of venereal affections in a very marked manner, as compared with their frequency in other parts of the country where they were not enforced.

With regard to the Navy, Dr. Dickson mentioned, in his presidential address to the Epidemiological Society in November 1885, that the last annual report of the Navy shows that, when the clause of the Acts regarding the compulsory examination of prostitutes ceased to be enforced in May 1883, the latter half of that year exhibited a very marked augmentation in the number of cases of syphilis, both primary and secondary, gonorrhœa, and epididymitis among the seamen and marines, averaging 22,000 men. The ratio of such cases for the year 1883 was 280 per 1,000; the number of men thereby disabled was 367 per diem, or 17 per mille of the force employed. The mean ratio of the previous ten years had been 132 per mille, and the whole number on the sick-list from these disorders had given a daily average of 10·6 men per mille. Sad as this state of things now is, the prospect before us is far more deplorable, seeing that, twenty-three years ago, when the adoption of the at present discountenanced system of prevention was beginning to be contemplated in England, Professor Maclean showed¹ that, in comparing the statistics of venereal diseases in the garrison of Paris, in 1852, with those of certain of our own, the admissions were: Paris, 24 per mille; Aldershot, 411; and Woolwich, 512. As I have been long out of India, and as I have no personal experience of the working of the system during the past twenty-four years, I am not qualified to offer an opinion upon the Indian data cited above, but I most earnestly trust that, in the highest interests of suffering humanity, the whole facts of the case, as it affects the character and working of Lock Hospitals in the East, may be dispassionately investigated and reported upon by military surgeons of great Indian experience. However the question may be finally decided, I shall never cease to believe that, whatever the unavoidable shortcomings of this system may be, *the direct tendency of Lock Hospitals, whether at home or abroad, is to reduce the prevalence and virulence of venereal disease, PROVIDED THAT THE INSTITUTIONS ARE JUDICIOUSLY AND LIBERALLY CONDUCTED.*

What the Indian Lock Hospitals needed in 1830-34, was reform, not abolition. The action of those who put them down was somewhat less rational than that of him who should deliberately prescribe a poisonous dose of prussic acid in a case of inveterate scabies. Can we hope that, despite the action which is being adopted in the United Kingdom, a wiser course may yet be taken in India?

¹ *Annals of Military and Naval Surgery* for 1863.

Miserable cases of destruction of the tissues of the mouth and jaws, resulting from the native practice of salivating by mercurial fumigation in syphilis, are frequently seen at our hospitals. Doubtless many victims, previously the subjects of malarial and scorbutic cachexia, perish in this manner outside. Either the patient inhales mercurial fumes from a *bhuttee*, made in the form of the cord of raw cotton inclosed in cloth used as a pipe-light, or he sits enveloped in a blanket, on a cane-bottomed chair, under which is placed a pan of ignited charcoal, upon which a native preparation of mercury, usually *rascapur*, a mixture of calomel and corrosive sublimate, is sprinkled. See the chapter on 'Diseases of the Jaws' in Fayrer's 'Observations,' p. 264, and my chapter on 'Diseases of the Mouth.' Dr. R. H. Stevens¹ narrates the case of a Bengali lad of 13, with spleen, in whom salivation by a hakeem caused sloughing of the lower eyelid and destruction of the eyeball, which came away almost without the use of the knife. He died exhausted.

HOSPITALISM—PYÆMIA—SEPTICÆMIA—OSTEO-MYELITIS.

In the year 1843, our knowledge of hospital disease, as it was incident on traumatic cases, was not extensive. When, in last century, one died while under treatment for an injury which was not, in itself, of a dangerous character, it was perhaps said that 'he had contracted a fever in the house, *which was a very common case; it might be from the foulness of the air in the house.*'² An old writer ascribed the frequent occurrence of pneumonia, in traumatic cases, to hypostasis, occasioned by long confinement in a recumbent position. Surgeons were only too familiar with tetanus, hospital gangrene, and erysipelas. Travers had written a work on 'Constitutional Irritation,' and James Arnott and Carswell had well described and depicted phlebitis as a sequel to amputation. When, in 1843, I published the result of a practical inquiry, made at Guy's Hospital, into 'Certain of the Causes of Death after Injuries and Surgical Operations in London Hospitals,'³ I had before me the observation by Dr. Thomas Hodgkin, that he had noticed mottling of the kidneys in two patients who had died after the operation of lithotomy, '*and in others who had sunk after operations and injuries;*' and a remark by Mr. Key, that he had scarcely ever seen a fatal case of lithotomy in which there was not discovered organic disease of some of the abdominal viscera, and more especially of the kidneys. These facts very nearly representing the state of our know-

¹ *I. M. G.*, vol. vii. p. 275.

² *London Journals for 1765 and 1777*, reports of cases in the Middlesex Hospital, which was then in a miserable state from want of funds.

³ *Guy's Hospital Reports*, 1843, p. 78.

ledge upon the subject of blood-poisoning in surgical cases 43 years ago—I showed, in my paper, that out of 153 fatal traumatic cases in Guy's Hospital, death was proved to have taken place from inflammation of secreting surfaces or internal organs (including the kidneys, liver, and spleen) in 134. In the remaining 19, the patients died from these causes: tetanus, sloughing, hæmorrhage, suppuration, erysipelas, and total deficiency of reparative action in the wound. In one case, the cause of death was not discovered. In the 134 cases in which the patients died of internal inflammations, there was marked disease of the kidneys, liver, or spleen, or of all those organs combined in 90. Upon these data, I urged the necessity of invariably taking the precaution of examining the state of the liver and spleen, and of testing the urine for albumen in each patient before deciding upon the performance of any surgical operation, and of postponing such operation, if possible, while either of these organs appeared to be enlarged or the slightest trace of albumen was detectable in the urine. I think that I may unhesitatingly declare that the above precautions were not employed in hospital when I recommended them, or until long afterwards. My researches received fair credit from Sir James Simpson;¹ but, in 43 years, they appear to have died of old age, surviving only in the writings of my friend Sir Joseph Fayrer. Side by side with that excellent authority, I observed this class of diseases in the Calcutta Medical College Hospital for nearly fifteen years. As I have mentioned elsewhere, this hospital was built for the express purpose of relieving the native hospital of its medical cases, the surgeons of the latter institution perceiving clearly that cases of dysentery and fever necessarily infect a surgical hospital. The new building was accordingly named the '*Fever Hospital*.' Had it been designated the '*Surgical Hospital*,' and had it been strictly set apart for traumatic cases, the result might probably have been more favourable; but, unhappily, the old plan of massing surgical and medical cases together in a common atmosphere was again adopted here; the result was that the place became a hot-bed for hospitalism in its direst forms.

Writing in 1854, when he was in the habit of visiting the wards of the still new building in his capacity of Government Examiner in the college, Dr. Kenneth Mackinnon stated that wounds made in operating there were apt to take on an erysipelatous action, tending to sloughing, especially in the rains. Subsequently to this, during the fifteen years of my incumbency as senior medical officer of that hospital, our best care could not keep its surgical wards free from hospitalism in its worst forms—pyæmia, osteo-mylitis, and, latterly, from 1873, erysipelas. Once, in 1866, there was a transient outbreak of hospital gangrene, which ceased when we removed the traumatic cases to tents on the flat

¹ *Obstetric Memoirs*, vol. ii. p. 10, and *Twelfth Clinical Lecture on the Diseases of Women*.

roof of the building. There were, however, few years, up to the time of my leaving, ten years ago, in which some of those operated upon did not succumb to gangrene. Full and most graphic accounts of the hospitalism of that very unfortunate building have been given by Sir Joseph Fayrer in his 'Clinical and Pathological Observations in India.' Within my recollection, the localities surrounding the Medical College and Native Hospital might have been regarded as vying with each other in presenting all the most hideous impurities which characterised Indian cities 30 years ago. The surgical and medical sick, cases of amputation and compound fracture, sloughing dysentery, and malignant cholera, occupied a common atmosphere. The natives treated there were poor, though not, for the most part, markedly scorbutic, but probably none were free from malarial cachexia. Although never swerving, practically, from their determination not to abandon this very showy building, the Government were liberal in carrying out most of our recommendations for its improvement. The eye patients had been removed to another building previous to my incumbency. The obstetric patients have, subsequently, been placed in a hospital of their own. At our solicitation, the native buildings which closely invested the hospital were cleared away, and the neighbouring town sewers and our own drainage were improved. We shared the benefits of the new water supply. The latrines, on the surgical side, were removed from the body of the building to separate towers. The surgical wards were, as far as possible, shut off from the medical. A lavatory and a separate ward for the male cholera patients were provided. The female cholera patients lay with the other sick up to the time of my leaving. We set apart a large and airy apartment (our council room and library) as an operation ward. We diminished the number of beds in the surgical wards, and laid down impervious floors throughout the building, which, as it now stands, has probably been made the best of; but I and its many able surgeons have not succeeded in convincing the Government that it can never, by any practicable means, be rendered a fit receptacle for the sick and wounded of a city, standing, as it does, upon an oozy basis of town filth, situated close to a huge malodorous public market, and with its beds lying in the course of the prevailing winds which blow stench from end to end of every ward.

In reply to my inquiry as to how the surgical patients had fared during the last eight years, my friend, Mr. S. Bowen Partridge informed me, in the middle of 1884, that of that period only 21 months, ending December 1879, were spent by him in surgical charge. During that time, there was no case in his wards of pyæmia, osteo-myelitis, or hospital gangrene. They were 'certainly freer from all forms of hospitalism than they had been, and their surgical operations were very fairly successful.' In those months, Mr. Partridge had 46 scrotal tumour cases with a mortality of five (one from erysipelas, of which there were several

cases. He had twenty lithotomy cases, with eighteen recoveries, one death (an old man who died from kidney mischief after the operation-wound had healed, and who was found to have a stone impacted in the ureter), and one who, having been recently operated on, was still in hospital when Mr. Partridge left. He used antiseptics very freely, and paid especial attention to the cleanliness of the instruments, and to this, in a great measure, he attributes the good result in the stone cases. He invariably dressed all large suppurating surfaces, such as scrotal tumour wounds, with carbolised oil, and an abundant outer covering of clean cotton-wool, which he looks upon as a most valuable antiseptic air-filter.

The following extract from Mr. Cockerill's Surgical Report of the Madras General Hospital for 1882 forms a pleasant contrast to my sad recollections of the Calcutta Medical College Hospital, although we never had to complain of want of ventilation there :—

‘All the surgical wards are cool and airy, being freely swept by the sea-breeze; and to this, and to the fact that the windows and doors are always open, I attribute the almost entire absence of erysipelas and other “Hospital Diseases.”’ In 1883, only three cases of erysipelas were treated. It is not clear that, in either case, the erysipelas originated in hospital.

Writing very lately in advocacy of Professor Gamgee's method of treating wounds by dry and infrequent dressings, Dr. Crombie, of Dacca,¹ observes: ‘During the past year, 197 “major operations” were performed in the Mitford Hospital here, with only one case of pyæmia . . . admitted from outside. No case originated in the hospital, and the students of the Dacca Medical School are dependent on such chance cases for examples of pyæmia. Osteo-myelitis is to me a mere name. It is many years since I saw a case of it. In proposing an amputation below the middle of the thigh for disease, the possibility of a fatal issue barely enters into the calculation.’ Undoubtedly this is a very great improvement upon the state of things which existed in the insanitary Medical College Hospital, Calcutta, in my time, when we despaired of recovery from amputations through the continuity of the femur. But, although I am a strong believer in the efficiency of the modern system of antiseptic dressing, I cannot think that science has yet achieved the permanent stamping out of ‘Hospitalism,’ or that it can ensure recovery from any considerable percentage of capital operations upon, say, gin-drinkers with advanced Bright's disease.

In Calcutta, osteo-myelitis took a long course of prevalence in the wards of my surgical colleagues, making its appearance suddenly and ceasing unexpectedly. By my friends' kindness, I watched nearly

¹ *I. M. G.*, March 1886.

every case. I regret that the following remarks did not appear while this form of disease was prevalent.

Can we, in amputation, find a covering of periosteum for the divided end of the bone, without the adoption of any sub-periosteal method?

I first proposed the following plan in 1875 :—

Some forty years ago, I dissected a stump which has, on many subsequent occasions, afforded me a good deal of interesting thought.

As a general rule, the soft parts, after amputation in the continuity of a bone, appear to be gifted with the power of healing kindly over the divided cancellated structure. When, in exceptional cases, this does not occur, the result is disintegrative suppuration, ending in necrosis, chiefly involving the cancelli of the divided bone.

This destructive form of inflammation has the still unexplained power of extending upwards to the cancelli of the bone beyond the next joint, as from humerus to scapula. I am not aware that, in any given outbreak of osteo-myelitis, we have any certain power of arresting the infection; still, I think that there is a conservative expedient to which we might always have recourse, and which deserves trial, on physiological grounds, whenever osteo-myelitis prevails. Further, I am strongly disposed to believe that, although it appears to be a retrogressive measure, the following provision could be made, with great advantage, in the majority of amputations.

In the case to which I refer, amputation had been made at the upper third of the leg. The patient had probably survived the operation about a month. The parts were greatly wasted. Anteriorly, there was a deficiency of integument, and the posterior flap was integumentary. The operator had not sawed off the sharp anterior edge of the tibia. This edge of bone was perfectly uncovered, but it was still living. Above, to the extent of about half-an-inch, and underneath to nearly the same distance, the soft parts had receded from the tibia, leaving an out-jutting right angle of denuded bone; still, although the soft parts had thus receded, or probably sloughed from its sharp edge, they had in no way deserted the tibia. Appearing to have considered that the flaps could not heal over a bony edge of such extreme sharpness, the soft parts had actively undertaken the task of removing this troublesome angle.

Above, in a semilunar line across the spine of the tibia, and below in a similar line crossing the cancelli and the cut edges of the bone, there extended along the margins of the remains of the anterior and posterior flap, fringes of granulations. This upper and lower row of granulations not only surrounded the exposed edge of bone, but had buried themselves in the osseous structure, and had grooved it so deeply as to render it evident that, had the patient lived a little longer, the two lines of granulations would have thrown off the angle of bone

(closing upon it as the cutting edges of a pair of bone pliers would do), and would then, doubtless, have met one another and thus have healed the stump. Place the knuckle of one bent index finger between the index and middle fingers of the other hand, then bring the joints of the two latter fingers together, and you will see the manner in which the two lines of granulations isolated the condemned angle of bone.

On most careful dissection, I found that the upper line or lip of bone-excavating granulation was a process into which the retracted edge of *periosteum* had expanded itself; and that the lower granulating line was, in a precisely similar manner, produced by the *fascia* of the integumental flap.

Above its upper granulating line, the *periosteum* was found quite healthy and firmly attached to the front of the tibia. Behind its line of granulations, the *fascia*, now acting as periosteum, was equally closely adherent to the cancellated structure.

These two very analogous structures were thus found equally co-operating in a salutary work, which, naturally as it appears to belong to the periosteum, could scarcely be expected from the fascia, unless upon a recognition of this interesting natural law.

When Aston Key first promulgated the doctrine that, in strumous arthritis, vascular granulation-like productions from the synovial membrane advance upon the articular cartilage, erode it in grooves, and so remove it, I paid close practical attention to the subject, and convinced myself, and (*pace* Mr. Bryant) have always remained satisfied of the truth of Key's observations, largely confirmed as they are by what we observe in the processes of chronic necrosis of bone shafts and caries. In this stump, a process, perfectly analogous to that described by Key, was employed by nature for the removal of the offending bone.

My father, a naval surgeon, amputated at Trafalgar (he had sixteen amputations in that battle), and on many other occasions. He was an insatiable reader, and had a most extensive knowledge of the professional history of his service from the middle of last century.

I wrote my paper upon the 'Causes of Death after Operations and Injuries in London Hospitals,' in his presence. I knew all that he knew about amputation. He feared tetanus and that malignant ulcer, which attacked all wounds in the septicæmic (some of his experience of this terrible disease is given at p. 488, vol. iii., of 'Trotter's Medicina Nautica'), but I am confident that he had never seen or heard of the condition of spreading bone inflammation which we now term osteo-myelitis. *In his time, all flaps were exclusively integumentary.* I venture to think that the above facts, proving as they do that fascia can perform the part of periosteum, plead for the re-adoption of integumental flaps, especially during the prevalence of osteo-myelitis and septicæmia.

HOSPITAL GANGRENE.

The chapter on Scorbutus should be read in connection with this chapter.

As an associate of scurvy, hospital gangrene has been long the subject of medical observation in India. The writings of Trotter are full of evidence of the fact that, for a considerable time after the introduction of lime-juice had freed the seamen of our Royal Navy from typical scorbutus, the men suffered greatly from sloughing ulcers—produced by slight causes, such as the contact of a drop of hot pitch with the skin—which were evidently of scorbutic origin. A scorbutic tendency is most extensively prevalent among the native population of India, and most especially in the provinces of Scinde and Behar; and scorbutus has repeatedly attacked our European-Indian forces in sieges, as at Lucknow, and in campaigns, as in the first Burmese war, when fresh vegetables and flesh meat became scarce. The writings of Hennen and other military surgeons show that malignant ulcer, of scorbutic origin, also committed great ravages among our troops in the Peninsular war. Consequently, unless the greatest care be taken to supply prisoners and detached parties of European and native troops with an adequate and sufficiently varied diet, scorbutus in its full development, and, even more dangerously, in its masked forms—malignant phagedænic ulcer, dysentery, &c.—may be expected to prevail among them. In 1818, Dr. J. Adam observed an outbreak of ‘an epidemic malignant ulcer, or hospital gangrene,’ which occurred in a body of native troops belonging to the Nerbudda Field Force. Only one European soldier was attacked, in consequence of the application of a blister to the side. Here the ulceration was moderate and recovery speedy. A boil, or a small ulcer, consequent upon a scratch or bruise, would cease to discharge pus; its granulations would become pale and slough rapidly, with an acute burning pain and extreme sensibility of the surrounding parts. Fever and great constitutional distress would set in, and the destruction of parts would extend equally in all directions, passing deeply into the cellular tissue and muscles. The colour of the sphacelated parts was a dusky grey. There was very little discharge. It appeared as if sudden death had seized the parts. Bone would be laid bare; there was great loss of soft parts, which had to be removed by scissors, and there was a tendency to alarming hæmorrhage. The general symptoms at this stage ‘were such as mark a high degree of the remittent fever of the natives.’ ‘In no instance did death ensue.’ ‘The disease resembled exactly what has been described to occur in the hospital gangrene of Europe.’ Dr. Adam observed that, on this occasion, the native soldiers suffered

greatly, while the European troops were almost free from the scourge, because the latter were well fed and clothed, while the former were very wretchedly accommodated and indifferently clad and nourished, not only the poor syces and grass-cutters, but also the higher classes of native soldiers, depriving themselves, with a view to saving, of many essentials of comfort, more especially in the field, where these are most required.¹

In 1824, Mr. J. Leslie witnessed an outbreak of 'gangrenous ulcer' in the 65th Regiment of Native Infantry at Prince of Wales's Island.² The disease, of which a very careful description is given, was hospital gangrene. There were 281 cases in eight months; of these 40 were fatal. The limb was amputated in nine cases, four of which were fatal. Among the ascertained causes, were an extremely meagre diet of rice and vegetables, usually of very inferior quality, insufficient covering, and overcrowding.

In June 1826, Superintending Surgeon Longstaff reported³ that 'intractable ulcers, of an erysipelatous and sloughing character,' had long been numerous in the hospitals at Benares and at others in that division. Common boils, on being laid open or bursting spontaneously, immediately degenerated into phagedænic ulcers. This tendency had existed for more than a twelvemonth.

Mr. J. C. Geddes published⁴ a very graphic account of 'malignant ulcer and hospital gangrene,' as he observed it, in the early part of 1827, at Prince of Wales's Island, where, as we have stated above, this malady prevailed among the sepoys in 1824. The disease was most severe and fatal. A common sympathetic bubo often degenerated into a phagedænic ulcer, occupying the whole extent of the groin, spreading to and laying bare the abdominal muscles, and then exposing the peritonæum; extending down the thigh and laying bare the artery, which was seen pulsating, but which displayed extraordinary power of resisting the ulcerative process, lying untouched in a mass of gangrenous matter until the patient sank exhausted. Amputation was freely resorted to. Mr. Geddes and Dr. Grant amputated thirty-five limbs, besides toes and fingers. Exactly half of those so treated recovered.

Some years later, there was great prevalence of phagedænic ulceration of a most severe and obstinate character among our native troops in Arracan, when the constitutions of the men had become broken by Arracan remittent fever. (*Vide* page 257.)

In 1843, the 4th Regiment Madras Native Infantry suffered considerably from sloughing ulcers at Singapore. Ephemeral and quotidian fevers and rheumatism were prevalent. Phagedænic ulcers began to appear in the month of September, and continued to increase

¹ *C. M. T.*, vol. iii. p. 132.

² *Ibid.*, p. 151.

³ *Ibid.*, p. 418.

⁴ *C. M. T.*, vol. vi. p. 147.

up to the end of November, when the cases became less numerous and severe. The worst description of ulcer was generally situated about the lower third of the leg, or on the ankle and foot, and usually proceeded from some slight injury or from the pustules of itch. The expression was anxious, the tongue white and flabby, and the lower limbs frequently somewhat œdematous. Several patients died, 'either sinking from the effects of constitutional irritation, or, being reduced to a state of excessive debility, were carried off by colliquative diarrhœa after the sloughs had separated.'¹

Kenneth Mackinnon remarked that the hospital gangrene which appeared among the wounded after the battles on the Sutlej, in the first Sikh campaign, offered a fine illustration of the manner in which the atmosphere may be poisoned by overcrowding and foul discharges not carefully removed. 'The military surgeon,' he said, 'should not overlook the fact that the canvas of tents is a substance to which impure effluvia are likely to attach themselves.' In the next campaign, things were better managed, and the disease did not appear.

At Chittoor (Southern Division of Arcot, Madras Presidency) sloughing ulceration prevailed to a great extent at particular periods, as in the years 1830, 1833, 1837, and 1838. It was supposed to arise from the causes which produce fever. '*The inhabitants of the village were as liable to this form of ulceration as the prisoners, and were affected precisely at the same periods.*' I emphasise this fact, because, when we place it beside the fact which will presently be noticed (p. 342), that sloughing phagedæna prevailed extensively in the district of Indore, Bombay Presidency, it appears nearly certain that this malady does, to say the least occasionally, assume an epidemic or widely spreading endemic character. In the jail, ulcers formed a large proportion of the admissions. Many of the sores sloughed. In all these cases, the general health was more or less disordered, many of the patients had occasional paroxysms of fever, others suffered from diarrhœa, and the tongue was generally thickly coated with yellow fur. In cases where the constitution had been severely injured by repeated attacks of fever, the ulcers, having degenerated into large sores, became covered with a thick slough of a dirty white colour, attached to an irritable bleeding surface, which, on falling off, exposed the muscles, and sometimes laid bare the bones of the leg (the lower extremities only being attacked) to a considerable extent. In these severe cases, diarrhœa was generally present.²

In and about the year 1833, hospital gangrene was very prevalent among native prisoners in Bengal; the exciting cause being frequently irritation by the ankle-irons, before the leather *mozas* were brought into general use as a protection to the skin. This, as well as the

¹ *Madras Medical Topographical Reports* (Eastern Settlements), p. 233.

Ibid. Centre Division), p. 56.

attendant dysentery, was attributed to scurvy. A valuable chapter on this subject will be found in James Hutchinson's 'Observations on the General and Medical Management of Indian Jails, &c.,' 1845. That and other old records of scurvy and gangrene in Indian jails deserve the fullest attention of medical officers at present serving in the East, seeing that the returns for 1881 show that 405 cases of scorbutus, and 115 of 'gangrene and phagedæna,' occurred among the jail population in that year. The deaths from scurvy were only 9; those from gangrene and phagedæna, 48, of which 31 out of 45 cases occurred in the Andamans and Nicobars; and that there were, in 1882, 445 cases, with 13 deaths, of scorbutus, and 31 cases of 'gangrene and phagedæna,' with 13 deaths, among the prisoners—strong evidence that the old evil is always ready to break out afresh. My friend, Dr. Thomas W. Wilson, tells me that, while this disease was one of the chief scourges of Bengal native prisons, the late Dr. Wilkie, then, I think, of Dinagepore, took him into his jail hospital, where, to his utter consternation, he being then inexperienced in Indian disease, upwards of a score of prisoners who had undergone amputation stood in a row, leaning upon crutches. In 1836, Mr. Chapman, of Purneah, wrote to the Medical and Physical Society of Calcutta upon the great prevalence of sloughing ulcers in his jail. The disease was clearly hospital gangrene.¹

In and about February 1838, it was reported² that, at Agra, a scorbutic taint existed among the sepoys; and that, whenever the skin got broken in any way, a malignant ulcer formed. At the same time, hospital gangrene appeared among the sepoys at Saugor, and the disease broke out in Brett's Surgical Hospital in Calcutta.

Kenneth Mackinnon wrote³ that he once witnessed a very severe invasion of hospital gangrene in the jail hospital at Tirhoot, at a time when the weather was extremely close and sultry after a rainy season, during which the fall of rain had been unusually copious. The hospital was encircled by stagnant water. The destructive process was not accompanied by much surrounding inflammatory action. It was what he might call an eating away of the textures, with highly foetid discharge from the sores. The countenance had a sodden look. The conjunctiva tinged, perhaps not with bile, but with diseased blood.

I have been favoured by Dr. John Craig with the perusal of reports which he sent to the Medical Board of Bombay upon 'Aden ulcer,' which, upon assuming the superintendency of the Medical Depot at that station in December 1856, he found to be prevailing there, especially in the hospital for biggaries [native labourers] and other natives,

¹ *I. M. J.*, vol. i., N. S., p. 677.

² *I. M. J.*, N. S., vol. iii. p. 177.

³ *I. A.*, No. 3, p. 178.

and which had continued to occur there from the year 1839, when Aden was captured. Dr. Craig shows clearly that the disease was hospital gangrene, occurring principally in coolies from Bombay, foreign to the place, and not always well selected as regards their previous health. Scurvy, attributable to the use of brackish water and an insufficient supply of fresh vegetables, predisposed these unfortunates to sloughing phagedæna upon their admission, with any kind of ulcer, as from guinea-worm, bubo, &c., to a damp, dark, and ill-ventilated hospital.

The means which were successfully adopted were removal of the sick to tents in a dry situation, and treatment of ulcers at the patients' own quarters; due attention to food-supply, and the employment of nitric acid in dressing.

Dr. J. P. Walker reported, also in 1856,¹ that hospital gangrene was, for some years, a terrible pest in the Agra Central Prison, 'affecting alike the weak and the strong, the sick and the healthy, the aged and the young, those who had spent years in prison and those but recently imprisoned. In 1853, when the total deaths, from all causes, were as 5·2 per cent. of the average strength, no less than 23·9 per cent., or nearly one fourth of the whole mortality, was caused by 41 fatal cases of hospital gangrene. Everything was in vain tried as a prophylactic, and as a remedy; hygienic measures on a grand scale, resulting in the most scrupulous cleanliness, were found of no avail. The affected were isolated, and removed to the country for change of air, and the airy upper-storied rooms of the lofty gateways of the royal gardens at Secundra were converted into a hospital. Every plan of treatment—medical, surgical, and dietetic—recommended in standard medical works, and everything that theory suggested, was tried in vain, the pest remained unstayed; and, in a large number of the worst cases, death alone arrested the fearful ravages of the disease, and soothed the agonised tortures of the patients, the very remembrance of which is painful.'²

When I joined the service in 1848, nothing was heard of hospital gangrene, either in sepoy lines or in native jails, in *Lower Bengal*. Doubtless, in the preceding decennium, sanitation had done much for both soldiers and criminals. Nevertheless, our experience of hospital gangrene in India did not then come to an end, as is shown in Dr. J. P. Walker's 'Agra Report' cited above, and in the Jail Statistics of 1881 and 1882, which I have quoted.

In 1881, 'gangrene and phagedæna' did not occur very frequently in the native army; but, wherever it appeared, the type of the malady was severe. In Bengal, there were four cases with one death; in Bombay, three cases with two deaths; and in Madras, five cases with

¹ *I. A.*, No. 9, p. 83.

² The mode of treatment successfully adopted by Dr. Walker will be cited below.

one death. In 1882 there were four cases, of which two were fatal, in the Bengal Presidency, and two cases (one fatal) elsewhere.

The old Chandney Native Hospital, Calcutta, was ill-situated in Durrumtollah. Some forty-five years ago, sloughing was so destructive there as to lead the authorities to erect the Medical College Hospital, which was originally intended to relieve the other of its medical cases. The new building ought to have been reserved for surgical cases, for the old one continued to be impested with hospital gangrene.

In 1854, Dr. K. Mackinnon alluded to the fact that wounds made in operating there were apt to take on an erysipelatous and sloughing action, especially during the rains. An investigation of the history of this place convinced me that, as far as I could trace back, *hospital gangrene had been endemic in this building*. That is to say—while it frequently happened that compound fractures, amputations, lithotomy cases, &c., all of which were very numerous there, often recovered as well and as rapidly as they could do in any hospital (the only drawback being that the scalp-wounds, which were extremely common, generally took on an erysipelatous character, ranging from a slight evanescent blush of inflammation down to most extensive sphacelus); on the other hand, suddenly, as it would appear from some change of wind or weather, the surgeon, on paying his morning or evening visit, found that nearly every unhealed surface—ulcers, stumps, wounds which, at the last visit, only presented a thin line of healthy granulation—had become overspread with a thick, tenacious, diphtheria-like exudation, betokening gangrene. Some of the least promising of these cases did remarkably well, but a large proportion were fatal. I obtained from my friend, Dr. Herbert Baillie, the statistics of the gangrene which prevailed there for a period of four years and a half—1855–59. There was a total of 519 surgical operations, in 102 of which the issue was fatal. Gangrene attacked 96 of the 519 persons operated on, and 70 of these 96 died. This table did not include the many cases in which gangrene attacked ulcers and accidental wounds.

In the winter of 1859, I was requested by my friend, the late Dr. Allan Webb, surgeon to the institution, to visit its wards daily for a month, and to report the result for the information of the governors. Dr. Webb informed me that what he considered to be true hospital gangrene had not made its appearance in his wards since the year 1855; but that, since he had held charge, there had been scarcely any time at which he had not found reason to fear that any case of wound or injury, whether slight or severe, or any patient upon whom a surgical operation had been performed, however favourable in appearance all concurrent circumstances might be, was liable suddenly to be attacked with mortification. On my first visit (on January 29), I saw thirty-one cases of wound or ulcer. Seven of these patients had undergone

surgical operations. In six of the thirty-one cases, the wound or ulcer had a more or less unhealthy character. The nature of these unfavourable cases, taken together with what I had learnt from Dr. Webb, prepared me to believe that, while the generality of the wounds did well, many were attended with unhealthy inflammation, tending to gangrene, for which the nature and severity of the accidents or operations were insufficient to account. On the evening of February 1, my attention was drawn by Dr. Webb to a sudden and remarkable change for the worse which came over several of the patients suffering from wounds. They became feverish, or had distinct attacks of intermittent or remittent fever, their wounds gaped, were raised and flabby at the edges, or began at once to slough. These portentous changes were, at first, most observable in certain cases of flap wound. Several deaths followed this outbreak, and several cases of rather extensive gangrene did well, although their recovery was not rapid. The case of a native man, who had been operated upon for strangulated hernia, particularly struck me; there was a clean firm linear cicatrix which would have been perfect had not one bright red granulation, like a little carnelian button, remained at the middle of its course. Just as the man was about to be discharged, there was an appearance as if the surface of the granulation had been delicately touched with cigar ash, it being coated with a fine greyish-white film. Then most destructive gangrene rapidly set in and spread.

The native hospital and its most insanitary neighbourhood were afterwards improved; but the place was, at last, condemned and disused, the Mayo Hospital having been built for its patients.

Assistant-Surgeon R. N. Roy, published¹ a report of an outbreak of hospital gangrene which occurred in the Allyghur Dispensary in March 1874. Whenever the hospital accommodation is close and bad in India, and the patients are the subjects of scorbutic or malarial cachexia, hospital gangrene may be anticipated.

We, however, have evidence that disease of this character may suddenly make its appearance and become very prevalent in a district apart from the influence of poor food and overcrowding. Dr. T. Beaumont reported² that sloughing phagedæna was almost unknown in Indore until the rainy season of 1871, when a few cases were seen. During the rainy season, and up to the end of 1872, it became so very prevalent that it was feared it was about to become established as an endemic disease. In the five months ending December 31, 315 cases came under observation; of these 51 proved fatal. Any part of the body was liable, usually the leg, foot, or ankle. In all cases, it attacked some pre-existing breach of surface, as a slight abrasion from an ill-fitting shoe. There was nothing exceptional in the season or food supply.

¹ *I. M. G.* for November 1874.

² *I. M. G.*, vol. viii. p. 318.

Prophylaxis and Treatment.—As it has been found, in the very large majority of outbreaks of hospital gangrene, that some or all of the following causes have been at work :— Scorbutus, or what amounts to the same thing, poverty ; an innutritious staple of diet, such as the new common *dasee* rice of Bengal ; a diet not sufficiently full and varied, and wanting in flesh, fish, oil, and antiscorbutic fruits and vegetables ; overcrowding, especially in hospitals where surgical and medical cases are not separated ; insanitary *entourage*, exposure to vicissitudes of temperature ; over-work or fatigue, all within the influence of marsh miasma :—our chief means of prophylaxis lie clearly before us. With prisoners, abrasion by the leg irons, and with soldiers, chafing by shoes and accoutrements, are especially to be avoided, and the, at first, trivial sores thus caused should always be carefully treated. I think that the histories cited above show that recourse to amputation is not to be wholly put aside—I need scarcely add that I should not have tried it until all other means had been tested and had failed. In the Tirhoot Jail Hospital, Kenneth Mackinnon ‘ found no local application do good but the pure undiluted nitric acid, applied on lint, making the parts smoke, and literally burning out the whole diseased surface.’ So, also, in the Indore outbreak of 1871, Dr. Beaumont found no treatment so successful as the thorough application of nitric acid. My friend, the late Dr. J. Sutherland, used *irrigation* with marked success ;¹ and Dr. J. P. Walker places the highest value upon the local application of burnt alum and of an ointment composed of powdered alum (not burnt), catechu, balot (extract of *Berberis lycium*), opium, common bazaar soap, and water.²

SCORBUTUS.

The chapter on Hospital Gangrene (p. 336) should be read in connection with this chapter.

Very nearly first among the truths to be recognised by the young medical man commencing practice in India is the fact, much insisted upon in these pages, that scorbutic and malarial cachexia most frequently exist combined in the constitutions of his European and native patients.

Scurvy is generally prevalent throughout extensive tracts of Hindustan, especially Scinde and Behar ; and, everywhere else in India, the influence of a scorbutic diathesis ought to be looked for in investigating the origin and nature even of those diseases which are not generally regarded as being allied to scurvy. In India and elsewhere, the

¹ *I. A.*, No. 8, p. 470.

² For minute directions as to the preparation of these medicaments, *vide I. A.*, No. 9, pp. 84, 85.

wealthy, who are well able to live generously, are by no means always exempt from scorbutic cachexia, easily recognisable by those intimate with the indicia of scurvy. At a meeting of the Epidemiological Society, held in December 1883, Dr. Colan, R.N., observed that, on his return from Nares's expedition, he observed, in England, cases of scorbutus which, but for his experience of Arctic scurvy, he would have failed to recognise. The prevalence of the taint of land scurvy in England has always been recognised, and that not merely among the poor and underfed. When I was a boy at Portsmouth, my father and other naval surgeons were consulted in the case of an elderly gentleman who lived in affluence, and had a famous apple-orchard at the back of his house. He was dying of a disease which the local practitioners had failed to discriminate, but which the naval men at once perceived to be scurvy, and cured with oranges. In India, many well-to-do people get out of the habit of eating fresh fruits and vegetables. During the rainy season, when dysentery and acute hepatitis are most prevalent, these are rare, and servants do not provide them except under pressure. In Calcutta, I once insisted, as a matter of experiment, that my butler should give me a fresh salad every day for a year, and he succeeded in doing so. Mustard and cress and water-cress are always available, and lettuces more than might be expected. Common and very ignorant prejudice leads many into scorbutus. 'Mangoes cause boils'—only because boils are generally prevalent in the mango season; 'fruit gives cholera'—any gross excess of diet being an exciting cause of that malady. I always had good fruit on my breakfast table, on principle. Again, the old Indian's appetite (not always a safe guide) generally inclines him rather to savoury flesh meats than to fruit and vegetables.

I have written so fully elsewhere upon Indian scurvy¹ that I must trust that the reader who desires to investigate the subject at large will refer to those pages. I have shown that if, with the map before us, we draw an imaginary line from Aden upwards, principally within and near the stretch of the Indian Desert, through Scinde to Mooltan, we roughly define a tract in which apparently very trifling injuries, abrasions, scalds, contusions, are liable to degenerate into ulcers of the most intractable character, and even into sloughing phagedæna, or hospital gangrene. Along this line, land scurvy is rife; but, as it appears that the malignant ulcer of the fleet, prevalent at the end of the last century, which is so fully described in Trotter's '*Medicina Nautica*' and which was recognised at the time as resembling ulcers prevalent in the East Indies, committed its direst ravages subsequently to the almost entire disappearance of sea scurvy, pure and simple, under the influence of lime-juice, so it is probable that these forms of ulcerative

¹ 'Review of the Means of Preserving the Health of European Soldiers in India,' *I. A.*, No. 13, for 1860, pp. 589-620.

disease are often merely congeneric to scurvy, seeing that they by no means readily yield to antiscorbutic treatment. Still I believe that it may be laid down as a certain law that the subjects of these ulcers invariably have a scorbutic taint; and that, consequently, antiscorbutics must always be steadily employed throughout the treatment, however obstinately the local disease may appear to resist their power.

Among these forms of ulcerative disease, the principal are known as the *Aden ulcer*, *Scindh boil*, *Gwalior ulcer*, and perhaps *Delhi boil* (which, however, the late Dr. Gavin Milroy and Dr. Murray have regarded as a species of yaws). Such forms of ulcer were also very prevalent, in association with scurvy, among our native troops during the first Burmese War in 1825. Similar ulcers have been observed as occupying a prominent place among the endemic diseases of Malacca, Penang, Singapore, Batavia, Amboyna, and the Molucca Islands, Ceylon, the Andamans, and the Malabar Coast. It may be taken as a general rule that, wherever these obstinate forms of ulceration have prevailed, syphilis and dysentery have been markedly severe, the patients being scorbutic, the former being liable to run into sloughing phagedæna, the latter often terminating as gangrenous colitis.

I have already spoken of the fact that scorbutus is generally to be anticipated and guarded against as one of the most incapacitating and destructive attendants of sieges and protracted campaigns in India. I know officers who, having become scorbutic in the second Burmese War, in 1852-53, and during the siege of Lucknow, have never completely shaken off the sequæ of this grave cachexia.

It is highly noteworthy that, while districts impested with the marsh poison afford a fitting habitat to scorbutus, malarial cachexia and scurvy are, if not allied conditions, states which most readily coalesce and run into one another—with double peril to their victims. I repeat that, clearly, the fevers and dysentery of sea-faring men, which formed the principal subjects of those ships' surgeons who wrote upon the 'Diseases of India' late in the last century, were greatly modified, as occurring chiefly in men whose constitutions were tainted with sea scurvy; and that it has been considered, with great reason, that the so-called 'ship dysentery' which prevailed most destructively in our fleets, during last century, was the enteric fever of Jenner, *plus* scorbutus.

More than forty years ago, the late Dr. H. H. Goodeve made some excellent observations upon this point, which still well deserve the attention of practitioners.¹ 'The melancholy accounts of the ravages committed by scurvy found in every narrative of a long sea voyage, even up to a very late period, are alone sufficient to account in a great measure for the frequent occurrence of that typhoid' [i.e. typhus-like],

¹ 'Sketch of the Progress of European Medicine in the East,' *C. M. T.*, vol. viii. part ii. p. 118 of Appendix.

‘or putrid type of fever which the old surgeons, and especially the naval ones, so much dreaded during their residence in warm climates. The miserable condition to which the sufferers were reduced by scurvy was precisely the most unfavourable state in which they could be placed to receive the attack of a severe febrile affection. The scorbutic diathesis required only the accession of a remittent to constitute the putrid synocha of our forefathers. We consequently find the most melancholy accounts of putrid fever in the East amongst the reports of our naval surgeons. This form of fever was not, however, confined to the sea or its shores; it existed occasionally upon land, and raged with great violence.’

I had much experience of sea scurvy in the crews, principally of American ships, entering the Port of Calcutta, up to 1866. In that year, Dr. L. H. Lees published, in the ‘Indian Medical Gazette,’ vol. i. p. 287, an account of this disease, numerous cases of the worst description of which, in his capacity as Superintendent of the Seamen’s Hospital at Akyab, he saw in vessels arriving from Aden, whither they had carried cargoes of coal from England, for the P. & O. Co.’s depôt. No vessels entering the port, with the exception of those which had called at Aden, had cases. ‘One vessel, the crew of which was so reduced as to be unable to work the ship, had to put back to Madras, send all the men ashore, re-engage a fresh crew, and proceed on the voyage. Four others arrived at Akyab with only the cabin occupants fit for work. As may be conceived, they with the utmost difficulty made the port at all, and I do not hesitate to say that a few days’ prolongation of the voyage would have been fatal to all concerned. The other vessels were all more or less crippled by the disease.’

In 1839, Mr. W. A. Green published¹ a valuable paper in which he gave cases of sea scurvy, of putro-dynamic fever, and of fever complicated with severe spleen disease, with remarks to point out severally a similarity in their probable causes, in their nature, and in some of their symptoms. Mr. Green wrote from his experience at Howrah, across the Hooghly, facing Calcutta. I was one of his successors in the medical charge of that station; it is a position in which the meeting and combination of cases of malarial splenic cachexia and of sea scurvy could best be observed when the latter disease was prevalent, as there are extensive docks, and as many seafaring men, Europeans and natives, then frequented and resided in the place. Mr. Green analyses 43 cases of ordinary sea scurvy, and 9 cases of scorbutic febrile disease, arguing that, between these two cachexiæ, there appears a great similarity as to their essential nature—I would say rather in several of their characteristics than in their ‘essential nature.’ You cannot give a well-fed man scorbutus by locating him in a marsh,

¹ *C. M. T.*, vol. viii. part ii., Appendix, p. 303.

any more than you can give another ague by giving him improper rations at sea.

Twenty years later, under improved arrangements for victualling and lodging the men of our merchant navy, cases of sea scurvy were far less common in the Port of Calcutta; but only too many (one of which was fatal) came under my notice while I was in charge of the Medical College Hospital from 1861 to 1876. Most of these were, as I have already mentioned, from foreign ships.

One of the most valid maxims which an old Indian physician can offer to a young one is: *Never treat a case of any severe disease in India, whether the patient be European or native—landsman or sailor—without taking at least a glance at the question, Is scorbutus possibly a factor in the causation of this malady?* This advice is not underserving the attention of home practitioners.

Writing, in his 'Pathologia Indica,' in 1844, the late Dr. Allan Webb, an admirable observer, insisted that, among the troops serving in Bengal, spleen disease and *consequent scorbutus* are a very frequent cause of invaliding and of death. So intimate, in his opinion, is the connection between certain morbid states of the spleen and the induction of general scurvy that he appeared to consider the latter as a necessary result of the former. He says: 'Now if the spleen be as intimately concerned in forming red blood, as I have with much labour endeavoured to prove that it is, we have only to stop its functions and we shall then produce scurvy. Thus scurvy is induced by disease of the formative organs of the blood-globules, as effectually as it can be, by withholding the material from which blood is formed, by feeding the animal man upon putrid meat and bad grain, and washing down such a *pabulum vitæ* with stinking, stagnant water. In either case, the blood is not renewed; the body therefore feeds upon itself.' Without literally following this eminent man's line of argument or expecting that removal of the spleen will induce scurvy, every one who has practised in India must agree with Mr. Green and him that scorbutus and splenic cachexia have many leading features in common, and that the two cachexiæ, although they are perfectly distinct entities, often prevail together in the constitutions of Indian patients.

Study of the history of Indian scurvy clearly evolves this axiom: *Scorbutus often gives deadliness to Indian fever; and Indian scurvy never rages more obstinately than among the subjects of malarious cachexia.*

The literature of *Land Scurvy* in India, as occurring principally among troops in the field and in garrison, and among native prisoners in gaols, is extensive.

In 1832 a markedly scorbutic condition prevailed among the European soldiers of H.M.'s 48th Regiment, stationed at Cannanore, Madras Presidency, where good vegetables were scarce and high-

priced.¹ In the following year, there was a disastrous prevalence of scurvy in H.M.'s 62nd Regiment, stationed at Bangalore and Masulipatam. There were 57 admissions and 9 deaths.'²

Mr. R. N. Burnard³ described cases, as occurring during the military operations in Arracan in 1825-26, of native soldiers suffering from dysentery and burning of the feet, in whom the application of blisters to the calves of the legs produced a livid, bruised, and ecchymosed appearance; and, in one patient, caused sores which led to rapid sphacelus. Dr. John Grant published⁴ an analysis of the earlier reports on scurvy. He said that he believed cases of indubitable scurvy were then landed in Calcutta from Arracan and Rangoon. Many of these sank at once, being exhausted even by the exertion of disembarkation. Dr. Waddell described⁵ scurvy as prevailing among our European and native troops in this Burmese War of 1825. The same general symptoms characterised the disease in Europeans and in natives: 'swollen, loose, and livid gums, with ulcerated or sloughing edges, foetid breath, pain and hardness of the calves of the legs, contractions of the hams, and purplish discoloration of the skin, particularly of the lower extremities. In many instances of Europeans, diarrhoea and œdema of the feet, ascites in one case and universal anasarca in another.' 'A scorbutic diathesis was marked in almost every case of dysentery.' In all the worst native cases, 'the feet became cedematous, and, however apparently mild the other symptoms, the œdema was an almost certain prognostic of anasarca, hydrothorax, and death.' [Beriberi?] There is a valuable report by Superintending Surgeon Heward on the prevalence of scurvy among the European and native troops engaged in this war.⁶ It was observed that 'the disease which so generally afflicted the European soldiers, and with such fatal visitation, was dysentery combined with a scorbutic taint of constitution.'

Ulcers became numerous, greatly more so among the natives than the Europeans. The smallest scratch or abrasion of skin degenerated into an ulcer, in a few cases among the sepoys passing into the condition of 'hospital ulcer.' The duties of the men in the field were very trying and fatiguing. The diet of the Europeans consisted entirely of salt provisions, with biscuits or rice, 'but without any admixture of vegetable food.' 'Little or no fruit was procurable at Rangoon, and the only vegetable available to the troops was wild spinach or country greens, and that even in a very limited quantity.' *Provision of lime-juice for the campaign appears never to have been dreamt of.*

¹ Dr. Henderson's *Madras Quarterly Journal of Medicine*, vol. iii. p. 324.

² *Ibid.* vol. i. p. 18.

³ *C. M. T.*, vol. iii. p. 25.

⁴ *I. M. J.*, N. S., vol. iii. p. 647.

⁵ *C. M. T.*, vol. iii. p. 240.

⁶ *Madras Quarterly Medical Journal*, vol. i. pp. 203-9.

Dr. Burt described the annual occurrence of land scurvy in the native lunatic asylum, Moorsshedabad.¹ In 1822, his attention was drawn to the disease in consequence of the occurrence of severe pytalism caused by a small dose of calomel given to a patient with slight fever. The disease was 'very insidious in its progress, and Dr. Burt was inclined to think that it might have occurred in previous seasons, and have been reported as fever or dysentery, these being the first symptoms of which the patient complains, *although his gums may have been in an ulcerated state for months before.*' In 1825, Mr. J. Hutchinson² saw cases of scurvy in Cachar. He described a fatal case as occurring there in a sepoy of the 7th N.I.

In 1834, Mr. D. Macnab published³ an account of scurvy as it occurred, in that and the preceding year, among the native troops at Nusserabad. No similar visitation had attacked the troops there during the previous fourteen years; but it seems to have prevailed there extensively in a cavalry corps about the year 1820. The people of the bazaars escaped, very few of the camp followers suffered.

Generally the disease was very intractable, nearly every mode of treatment which the surgeon's imagination could suggest (with the exception of antiscorbutics, the idea of lime-juice never having apparently occurred), including large bleedings, having been tried. In describing the disease as it then occurred in the 4th Regiment of Light Cavalry, at Nusserabad, Mr. T. Ross⁴ says that he found medicine almost unavailing, although 'mineral and vegetable acids were liberally employed.' It was then usual to give nitrated vinegar in scurvy, but the report does not mention lime-juice. Most of the fatal cases sank from diarrhoea. In July 1837, Mr. Macnab laid another communication on scurvy before the Medical and Physical Society,⁵ strongly recommending, as an antiscorbutic, the fruit of the Anola, *Phyllanthus emblica*, *Emblie myrobalons*. Dr. Waring, 'Pharmacopœia of India,' describes this fruit as acidulous, purgative, and astringent, being employed in tanning. This authority cites Dr. Irvine ('Medical Topography of Ajmeer,' p. 118) as being of opinion that it does not possess any peculiar antiscorbutic virtues, and that, in this respect, it is not superior to any other acid vegetable astringent.

Dr. R. H. Irvine says that, when the disease described by Mr. Macnab (which occurred as a severe epidemic in the district of Ajmeer) appeared, bad grain was considered to be the cause, but he considered that facts did not seem to warrant this opinion. 'The disease subsided as the season became milder.'

In 1837-38, a great deal of scorbutic cachexia existed among the native troops at Agra.⁶ Dr. W. L. Macgregor showed⁷ that, in 1841-

¹ *C. M. T.*, vol. iv. p. 14.

² *Ibid.* vol. vii. part ii. p. 389.

³ *Ibid.* vol. viii. part i. p. 101.

⁴ *Ibid.* p. 130.

⁵ *Ibid.* vol. viii. part ii. p. 201 of Appendix.

⁶ *C. M. T.*, vol. viii. part ii. p. 282 of Appendix.

⁷ *Op. cit.* p. 178.

42, at Kurnaul, sloughing followed the application of blisters and leeches in the hospital of H.M.'s 3rd Buffs. This place appears to have been overcrowded, to have had a low verandah and an insufficient area, pent in by lofty buildings. The reporter adds, however, that scurvy appeared in other hospitals at Kurnaul during the same season. One of the most severe cases he ever witnessed occurred in a havildar of the 3rd Cavalry on whom a blister had been applied to the nape of the neck, and the whole of the integuments and adipose substance were destroyed by the sloughing, laying the muscles completely bare. We may infer from the context that the soldiers had an abundance of excellent food. We thus see that—say between 1820 and 1842—scorbutus prevailed at Nusserabad in the W. and Cachar in the E.; at Kurnaul and Agra in the N.W.; in Assam in the N.E.,¹ and Arracan and Burmah in the S.E. Are we to consider that this widely ranging disease was a true epidemic? It apparently depended much upon the prevalence of dry seasons, causing augmentation of the price of grain, a valid source of disease among parsimonious Hindu sepoys, who were the principal sufferers, serving in the field, or stationed in places distant from their homes, but a circumstance which could only have affected the European soldiers by diminishing their supply of fresh vegetables and fruits. The question of the epidemicity of scurvy will be continued presently.

An account² of the presumed remarkable efficacy of *free blood-letting* in sea and land scurvy, by Deputy-Inspector General Murray, Surgeon Bailey, R.N., and Assistant-Surgeon Armstrong, of the 29th Regiment, deserves to be consulted as a curious document, illustrating the 'good in everything' in therapeutics. A considerable amount of information regarding scurvy as it formerly prevailed in Indian jails will be found in my chapter on Hospital Gangrene.

In his admirable 'Medical Topography of Scindh,' the late Mr. Kinloch Kirk showed that the great characteristic of the Desert Tract, especially of Scindh, is its impregnation with saline matter, similar to what is obtained by the evaporation of sea-water. Everywhere that a well is dug the water is brackish and bitter. Throughout the Desert Tract, true scurvy is constantly endemic among the poorer natives, and there European troops have always been liable to scurvy, both in its masked and in its developed forms. There also scorbutic dysentery is a leading disease. Here it is noteworthy that, throughout the true Delta of the Ganges, the well water is brackish and undrinkable. Undoubtedly there is a considerable amount of scurvy in that immense tract; but, there, the disease is far less generally prevalent than it is in Scindh or Behar. The natives of the Delta use either river water or rain water, collected in more or less well-puddled tanks. One of

¹ Dr. McCosh at a discussion of Medical and Physical Society of Calcutta, July 1837.

² *I. M. J. N. S.*, vol. iii. p. 651.

the most important accounts of land scurvy in our Indian literature has been given by my friend Mr. Alexander Grant, in his excellent 'Medical Sketches of the Expedition to China,' in 1840 (Finch's 'Indian Journal of Medical and Physical Science,' 1845). While stationed on the north side of Hong Kong, the 37th Madras Native Infantry suffered much from fever, dysentery, and malignant ulcers on the lower extremities. These unfortunate troops, ill-fed, being debarred by caste prejudice from eating much of such limited supplies of food as were available, and ill-lodged in tents and mat huts, which a hurricane finally levelled to the ground, became, Mr. Grant informs me, thoroughly scorbutic, and then sloughing of the cornea was rife amongst them. At the same time, the Bengal European troops, similarly exposed and rationed scantily on weevilly biscuits and salt meat, were attacked with scurvy, bloated countenances, spongy gums, and legs swollen and covered with petechiæ, and often died suddenly from effusion into the cavities [beriberi?]. Ulcer was not developed in the European force, but cases of scorbutus occurred among the seamen in the ships.

In 1851, there appeared, in Mr. Woodcock's Prison Report, Appendix E., p. 150, a report by Mr. S. Clarke on scurvy in the Humeerpore Jail, and at about the same time¹ Dr. A. H. Leith reported the frequency of scurvy among the prisoners confined in the Bombay House of Correction, evidently in consequence of the absence of antiscorbutic ingredients in their food. The sickness was arrested by issuing a supply of *kokum*, the dried fleshy part of the *Garcinea purpurea*, a tree which is cultivated in the Konkan.

Having seen a notice, I think in Dr. Mouat's Jail Reports, of the prevalence of scorbutus in Behar, a by no means infertile country, I, on inspecting the principal jails of that district in 1865, examined every prisoner, and found a purple or dirty lake-coloured line edging the gums (quite like the lead line, except in its colour) in the great majority of the prisoners, numbering many hundreds.

At Dinapore, in that district, hæmorrhagic dysentery used to kill our European soldiers almost like a pestilence; and, while I knew the country, it continued to be very prevalent in the jails.

I often, but by no means so generally, found this indubitable sign of scurvy in poor Bengalis, as I have mentioned in the chapter on hospital gangrene.

Scurvy was, in old times, one of the direst scourges of Indian native jails. It was considered that, although probably more regularly fed, and at a greater cost than their brethren outside (when their food was not stolen by jail servants), the prison dietaries fell short of the requirements of health in not being sufficiently varied,

¹ *Transactions of the Medical and Physical Society of Bombay*, No. 1, N. S., for 1851-52.

and especially in not containing a great number of spices and of anti-scorbutic vegetables, such as *sag* (wild spinach), *mohwa* flowers, and cresses found in native gardens, and picked up in waste places, and by brooks and tanks. The wise measure of establishing a garden for each Bengal jail, carried out by Dr. Mouat, having been for many years in operation, the danger of scurvy among the prisoners ought, by this time, to be greatly diminished; but now, as always, scurvy is an evil which appears to be ever ready to show itself in India. In December 1868, we in Calcutta were surprised to see it reported¹ that an outbreak of scurvy had occurred in a fine Sikh regiment in Fort William. More than fifty were admitted to hospital, of whom five or six died, independent of twenty or more sent to their homes on medical certificate. The appearance of this disease was attributed to want of milk and butcher's meat, confined barracks, and protracted residence in an uncongenial climate. (*Vide* Dr. R. Temple Wright's 'Account of Scurvy in the Afghan War of 1878-81, and in the Jails of the Central Provinces in and before 1881.'—*I. M. G.*, Feb. 1886.)

The S. C. R. give the following statistics of scorbutus in India. In 1880, 41 European soldiers were admitted, 1 died. Cases did not occur among the women and children. There were 1,149 cases, with 32 deaths, in the native army. The prisoners had 467 admissions, and 8 deaths. In 1881, the admissions of European soldiers were 33, without any deaths. One soldier's wife and 3 children were admitted. There were 903 sepoys admitted, with 27 deaths. The number of prisoners treated was 411, of whom 9 died. In 1882, the admissions of European soldiers were 26. The women and children were free from the disease. Of the sepoys, 471 were admitted, and 18 died. Among the prisoners, 362 were treated, with 6 deaths.

Carefully read, these statistics are full of invaluable suggestions. Thus the occurrence, annually, of from 30 to 40 cases of scurvy in our force of some 60,000 European troops proves that the men are never quite exempt from the tendency to scurvy which prevails in certain Indian localities, and that this spark of disease, which now smoulders, will burst into flame at any moment in the field, on foreign service, in sieges, or whenever the troops may be deprived of their due supply of fruit and fresh vegetables. It will be borne in mind that from 30 to 40 *admissions* per annum in such a body of men evidences a very considerable prevalence of scorbutic cachexia among those whose condition of disease is not so pronounced as to compel them to seek relief in hospital. The annual occurrence of from 400 to upwards of 1,000 cases of scurvy among the 115,000 men of the native army shows that, although it does not rest with us to diet these sepoys, it is a matter of great importance everywhere, whether in cantonments, in stations remote from their own districts,

¹ *I. M. G.* for 1868, p. 283,

or in the field, and most especially in times of scarcity, that we should provide that abundance of good food, at moderate rates, shall be obtainable in their bazaars. The occurrence of a considerable amount of scurvy among prisoners is evidence that the jail garden system needs extension. It is probable that very little of the scorbutus which occurs among prisoners originates in the jails. The fact that, in the three years 1880-82, only five fatal cases of scurvy occurred in the jails of the Bengal Presidency, which, in the last of those years, contained 59,489 prisoners, a large number of whom came from districts where scorbutus is constantly endemic, is evidence that the jail garden system is working efficiently in that presidency.

It appears to me that, carefully weighed, the whole of our facts regarding scorbutus and hospital gangrene in India may be viewed as more or less directly substantiating the truth of Kenneth Mackinnon's opinion that *land scurvy assumes an epidemic character in that country*. In one of his last writings,¹ that excellent observer remarked: 'As far as my knowledge goes, in no other country has it been observed that land scurvy shows itself in an epidemic form, but it does so here, and particularly in Rajpootanah, and, as I think, also in Scinde. It was a knowledge of this which induced me, when a member of a committee directed by Government to report upon the probable causes of scurvy among the troops which came from England in the end of 1853, to give it as my opinion that the appearance of the disease on board so many ships, many of which vessels had, on previous voyages, brought troops in perfect health to Bengal, that the prevalence of scurvy' [*sic*] 'depended upon an epidemic constitution of the atmosphere in that particular season. The presence of scurvy on board of ships is usually assigned to the continued use of salt provisions; there was evidence before us that the voyages had not been of inordinate length, and that the men had been freely supplied with fresh hermetically sealed meat and with potatoes prepared in the same manner. In fact, the salt meat theory, as entirely true, must be abandoned, for the Rajpootanah epidemic was among regiments of Native Infantry where no animal food is consumed, and I am inclined to believe that epidemic scurvy may be independent of every kind of diet, for these sepoys in Rajpootanah used the same grain with the neighbouring villagers, who had no scurvy among them. In Scinde, and even at Lahore, a great prevalence of scurvy has been seen, with no changes in diet to account for it; and this is true also of the hills, as pointed out by Dr. Grant in his paper on hill diarrhœa.² The disease is also endemic in the jails of the Saugor and Nerbudda territories. The whole question is very mysterious, for while in the hills and on the voyage round the Cape in the year I have spoken of,

¹ 'The Epidemics of Bengal and N.W. Provinces,' *I. A.*, No. 7, p. 159.

² *I. A.*, No. 1.

unusual damp might be held to influence the presence of disease, in Rajpootanah, on the other hand, when the epidemic prevailed in 1835, the season had been very unusually dry; and, strange to say, in Rajpootanah the disease was confined to sepoys.'

The above appeared in 1857. In like manner I have shown, p. 338, that, in the years 1830-33, 1837-38, hospital gangrene, the congener if not the offspring of scorbutus, affected alike the inhabitants of the village of Chittoor (South Division of Arcot, Madras Presidency), and the prisoners in the jail, and both classes were attacked precisely at the same period. Again, I have cited (at p. 342) Dr. Beaumont's report to the effect that, Indore having scarcely known this form of disease, sloughing phagedæna became so very prevalent, during the rainy season and up to the end of 1872, that it was feared it was about to become established as an endemic disease. In five months, there were 315 cases, with 51 deaths. It is particularly noticed that 'there was nothing exceptional in the season or food supply.' (See also Irvine on epidemic scurvy in Ajmere, p. 349).

The opinion that scorbutus has, from time to time, appeared as an epidemic, rests upon a considerable mass of evidence. We read in Hecker that it did so, more especially in Germany, in the year 1346, and with such severe and unusual symptoms that people were inclined to regard it as an entirely new malady. Hecker cites various authorities in evidence that, on several occasions in the fifteenth and sixteenth centuries, scurvy occurred as an epidemic in Europe.

The subject of land scurvy in India deserves a volume to itself. I have treated it concisely, but I think that I have given, for those who may desire to study the subject fully, data which will enable them to refer to all the best authorities.

Note on Scorbutic Dysentery.—The very deadly sloughing and hæmorrhagic dysentery which prevails in many districts of India ought rather to be called scorbutus than dysentery—scurvy killing its victim at the large intestine. Although it should have a fair trial in every case, ipecacuanha, the sovereign specific in common dysentery, does not appear to have the power of touching gangrene of the colon, in the causation of which scorbutus is often, if not always, an element. I have shown, in the chapter on dysentery, that this scorbutic form of the disease is a gangrenous colitis which destroys life in about ten days—the colon lying, sphacelated throughout all its structures, dead among the normal small intestines. I have already expressed doubt as to whether this is, in reality, dysentery; but it usually commences with dysenteric symptoms. Ill-fed as the poor in Lower Bengal generally are, evidences of scorbutus, although frequently present, are by no means so common among them as in Scinde, Behar, &c. Lime-juice does not cure their dysentery; ipecacuanha often does. In Scinde, where indigenous fruit and vegetables do not abound, the prevailing

dysentery has, from old times, been treated antiscorbutically with good effect. Acting upon this fact, I determined, many years ago, that I would not let any dysenteric patient die without giving lime-juice a fair trial. I appeared, at first, to have some success; but, in Calcutta, probably from the comparative absence of scurvy, lime-juice was often powerless. When a man is admitted with a dead colon, you cannot hope to cure him with lime-juice. The patients like it, it never does harm, and, as the scorbutic taint may be latent in any case, especially in sailors, it is evidently a therapeutic agent which must not be overlooked.

Anæmia.—See 'Malarial Cachexiæ,' pp. 244 and 253.

Anasarca.—See 'Malarial Cachexiæ,' p. 251.

Beriberi Fever.—See p. 60.

TUMOURS.

Of *Tumours* generally, as they occur in India, it is observable that where, as frequently happened in old times when the skill of European surgeons was less believed in than it now is, growths, especially the non-malignant, often attained enormous bulk. This was very graphically shown in a series of pictures of Chinese tumours which used to hang from the gallery in Guy's museum. Apart from scrotal tumours, many of these vast growths are pictorially represented in the Indian medical publications of fifty years ago. A case, described and pictured by Dr. A. Campbell,¹ is especially remarkable. A Nepaulese lady of high rank had a massive growth originating above the right breast. Its size and weight were such that she could only move by the assistance of her slaves. It had descended so low as to rest on her couch when she sat upright. The continued pressure of its base on the ground or bed brought on fatal ulceration with fever. I removed larger fatty tumours in Bengal than I ever met with in England. I saw my friend, the late Dr. Allan Webb, remove an enormous tumour from the outer triangle of the neck of a native, the position and loose attachments of which suggested his remark that, when bulky tumours, originally deep-seated, are dependent and not malignant, long neglect of surgical aid often has the advantage that their weight causes them to enucleate themselves; and thus their excision becomes easy and safe.

Fibrous Tumours (*vide* Diseases of the Uterus) and most other non-malignant growths are not rare in India, and are fairly illustrated in the Calcutta Medical College Museum.

I only saw two genuine cases of *Hodgkin's Disease*, in a respectable European woman, in whom the progress of the malady was not at all

¹ *I. M. J.*, vol. i. p. 49, N. S.

active while she was under my care ; and in a native gentleman of high rank, in whom it proved rapidly fatal. In August 1880, Assistant-Surgeon Koilas Chandra Banerjea brought before the Calcutta Medical Society an elaborately but very imperfectly detailed case of a Moham medan, *æt.* 30, who died with an enormous amount of glandular disease (lymphadenoma) after about twenty-two months' suffering. It appears, although the report is very indefinite on this point, that a large aggregation of diseased mesenteric and other glands, matting together most of the upper abdominal viscera, had 'caused erosion of the vertebræ.'

Three preparations in the C. M., 242 to 244, illustrate *Vascular Tumours*.

Nævus.—Speaking of *Injuries to the Skull*, I have mentioned a case in which most extensive vascular nævi occupied nearly the whole of the scalp and the thickness of the calvarium in a Cookie girl. (See Diseases of the Skin.)

Molluscum fibrosum occasionally produces extreme deformity in natives. A by no means very unhealthy oldish man living near my college, had his body thickly covered by the characteristic pendulous tumours of all sizes up to that of a goose egg, and the growths on his head had produced such an extraordinary cowl-like elongation of the skin that it was widely pendulous, resembling in form the hanging portion of a cock's comb.

Dr. John Adam described and figured ¹ the case of a Hindoo, of Jessore, the subject of molluscum. His face and limbs and body generally were studded over with small berry-like sessile elevations, and a tumour, in colour and texture not unlike the skin of a toad's back, arose from the integument surrounding the ear, and, becoming very bulky, hung down over the neck in longitudinal folds to the middle of the pectoral muscle. The paper, which includes a report from a Hukeem, upon the native opinions regarding molluscum, should be consulted by those who are interested in this disease.

Mr. Brett reported and pictured ² the case of a native of Delhi whose body and limbs were covered with tumours of molluscum from the size of a pumpkin to that of a pea. Nine hundred and ten were counted. Mr. Brett removed one, weighing about three pounds, from the back. In June 1880, Mr. O'Connell Raye exhibited, at the Calcutta Medical Society, a native woman, *æt.* 22, who was born with a small nodule on the inner and upper aspect of her left armpit. Gradually increasing and becoming pendulous, it weighed seven pounds when Dr. Raye removed it. The whole of her body was covered with small nodular growths. Her mother had similar growths all over her body. Dr. Raye observed that the condition was hereditary and congenital. He had seen a similar case operated on by Dr. Farrell, in Dublin. Dr.

¹ C. M. T., vol. i. p. 293.

² I. M. J., vol. iv. p. 383, N. S.

McLeod drew attention to several cases of a similar kind described by Dr. James Wise, late of Dacca, published in a compilation by Drs. Fox and Farquhar on certain skin and other diseases of India, &c. Several of Dr. Wise's cases, and one contributed by Dr. Bainbridge, of Dholia, resembled very closely Dr. Raye's case. In three of Dr. Wise's nine cases, the disease was hereditary.

Dr. Chatham Gray¹ had under his care a native boy of 12, covered with molluscum, in whom the eyelids were especially affected. One eye was lost, the other was almost disorganised.

The work done by natives does not often make them liable to *Bursal Tumours*; but these may occur. One of the first voyagers in that sea noted in his log that two black devils, riding on fish, appeared in the Madras Roads. These were catamaran men, who, seated on a boat-shaped mass of timber, paddle about very dexterously. Prep. 98, page 147 of Sibthorpe's Catalogue is an enlarged bursa, of two years' standing, removed from the outer side of the foot of a fisherman, *æt.* 40, whose feet were constantly subject to irritation from pressure when rowing a catamaran. There was a similar hypertrophy on the other side of the foot. The walls were thick and uneven within with ridges and projections. It contained a clear yellow fluid in which numerous shreds were floating.

Cheloid.—Preparation 200 in our C. M. is 'A keloid outgrowth about four inches long, two inches broad, and one inch thick, formed in the cicatrix of a burn. The contrast between the whole surface of the growth, and the small portion of integument which has been removed with it, is very striking. It consists of fibrocellular structure. At Chittagong, a boy of 12 had the forearm drawn up by a burn cicatrix, the thick web of which was nearly a foot long and five inches deep. An extensive cheloid growth occupied the web midway, and was inclined to ulcerate. I removed a large triangular mass and straightened the limb.

A chapter, p. 552, of Fayrer's 'Clinical and Pathological Observations in India,' is devoted to a series of important cases of 'cicatrices after burns treated by surgical operation.'

In a report on the diseases of Kashmir,² Surgeon-Major Ross says that *Lupus* is common there. One desperate operation was performed for its cure—dissecting off the whole cheek and integument over the malar bone of a boy—successfully. Some cases were treated by nitric acid.

I never happened to meet with a case of lupus, not of syphilitic origin, in that country either in the European or in the native. Among 7,125 dispensary cases treated in Calcutta, Dr. Chuckerbutty met with only one example of lupus, which may have been syphilitic. Among

¹ *I. M. G.*, August 1878.

² *I. M. G.* for October 1878. .

1,920 cases of skin disease in natives which he treated in Calcutta, my friend Dr. William Palmer met with eight cases of lupus.¹ He draws a very clear distinction between the non-specific and syphilitic forms of lupus. He does not expressly define the character of these eight cases ; but, in a recent communication, he tells me that, in his experience, the non-specific is by far the more frequent type of this disease in Bengal. If it be true that the non-specific disease is a form of struma, its rarity may be due to the great immunity from the external forms of scrofula of the vast majority of the natives of the plains of India.

For **Tubercle and Struma** see *Phthisis*.

CANCER.

Forty years ago, Mr. Thomas Wilkinson King drew the following results from 1,000 *post mortem* examinations performed at Guy's Hospital.² 'Of all females who die at about 44 years of age, nearly one-half have cancers ; of males one-eighth. Of males above 65, one-fifth of all who die are cancerous.'

In 1884, Dr. Hofmeier, of Berlin, showed³ that, among 10,000 adult women admitted to the Elizabeth Hospital, in 1865-80, there were 358 cases of cancer.

Only too many cases of malignant disease present themselves in India, especially in the large city hospitals ; but, starting with the above facts, we must, I think, consider that the diseases of this class are by no means remarkably prevalent in India.

It is true that the great majority of soldiers' wives in India have not reached the cancer age. Still there are many middle-aged women among them. In the four years ending 1883, there were only six cases of cancer in an average strength of 3,676.

Although I have known it occur in several cases, true *Scirrhus* is undoubtedly rare in Lower Bengal. It is not, in India, the scourge of Englishwomen as it is at home ; while native women have the advantages of bathing regularly, of not wearing stays, and of being exempt from the noxious action of coal smoke.

Preps. 263 to 274 in the Calcutta M. C. Museum, well illustrate scirrhus. There is a great deal of *Medullary Cancer*, especially among the young of the native poor. I have seen enormous growths, chiefly of the hæmatoid form, repeatedly.

Epithelial Cancer is, I believe, the form of malignant disease most prevalent in India, and is probably quite as common there as it is in England, attacking the conjunctiva, skin, lip, tongue, os uteri, penis,

¹ *Rough Notes on the Common Forms of Skin Disease*, p. 25.

² *London Medical Gazette* for 1845, p. 597.

³ *Berlin. Klin. Woch.* and *M. T.*

&c., in Europeans, Eurasians, and natives. Two of my medical brother officers died from epithelioma of the tongue, a third and an officer's wife died from this disease attacking the glottis. I saw a very marked case of epithelioma of the penis in a pariah dog. The surgical report shows that, in the Madras General Hospital, there were fourteen operations for cancerous disease in 1882. Eight were cases of amputation of the penis for epithelial cancer. Five were for the removal of epitheliomata of the lower jaw, lower lip, tongue and submaxillary gland. In one case, the cancer of the penis was encephaloid.

Dr. Elmslie gave¹ a very remarkable account of epithelioma as it occurs among the native Kashmiri poor at Sirinagar. Out of 5,080 dispensary patients, no fewer than 30, or one in every 254 patients, were found to have unmistakable epithelioma.²

The disease, as it occurs in that locality, shows a remarkable preference for the abdomen and inner aspects of the thighs. It invariably occupied these sites in the 30 patients. Dr. Elmslie attributed the disease to the universal employment, in cold weather, of the *kangri*, an earthenware pot usually covered with fine wicker-work, containing ignited charcoal, which is worn under a loose woollen gown in close proximity to the skin. Indoors or in the sitting posture, the *kangri* is placed between the thighs. He suggested that the heat of this earthen vessel might act as that of a short-stalked tobacco pipe does in causing epithelioma of the lower lip.

Surgeon-Major G. C. Ross has confirmed the above observations as regards the cause and prevalence of epithelioma in Kashmir.³

At pp. 558 and 566 of his 'Clinical and Pathological Observations in India,' Sir Joseph Fayrer has some valuable remarks upon epitheliomata as occurring in old burn cicatrices exposed to irritation.

Preps. 251 to 252 in the C. M. well illustrate epithelioma in Europeans and natives.

I never saw a case of *Rodent Ulcer* in India. My colleague, Dr.

¹ *I. M. G.*, vol. i. p. 325.

² Without questioning the fact that epithelioma is very prevalent in Sirinagar, I may allude to a point which has often come practically to my notice—that, in India, as elsewhere, a fair amount of success in even a few cases of operative surgery often gains us a local reputation which leads a majority of those similarly afflicted to come to us even from a considerable distance. Hence, we may fall into the error of supposing that the disease in question is more prevalent, especially in our locality, than it really is. Thus, in the time of Esdaile and his successors, the dispensary at Hooghly had, beyond all others in Lower Bengal, a reputation for the safe removal of scrotal tumours. I often said, when a passed student told me that he had been appointed to a dispensary: 'Do not lose a month in attracting, by every practicable honest means, those who are known to all the town as having ugly but benign wens and fatty tumours. The successful removal of a few of these will establish your reputation more securely than years of undemonstrative medical practice will do.'

³ *I. M. G.*, October 1878.

William Palmer, tells me that, after I left, he treated three very well-marked cases of this disease in the Medical College Hospital. The patients were native men, all over 40 years of age. The nose was principally involved, and, the disease not being extremely advanced, cicatrisation was obtained by the use (under chloroform) of chloride of zinc.

The four years' returns, ending 1883, give only one admission for Rodent Ulcer, in a native prisoner.

As in England, *Melanotic Cancer* is an ordinary cause of death in Bengal among grey and white horses. We can scarcely drive through Calcutta without seeing animals having the characteristic globular tumours beneath the skin.

We are told, in the account of Bangalore in the 'Madras Medical Topographical Reports' (p. 70), that horses at that station are liable to 'enlargement of the tail, which becomes covered with large black tubercles constantly increasing in size and number; the tail becomes enormously enlarged, and, as the disease extends, the animal pines and dies. It appears to be of a melanotic character, and, on dissection, a black secretion has been found in the lungs, liver, kidneys, and in other viscera; and, in one case, a melanotic spot was discovered in the heart.' In India, as in England, this form of cancer is rare in human subjects. I saw what appeared to be the commencement of such deposit on the foot of a well-to-do East Indian. It has occurred in the eye-ball,¹ and on the hand.²

Dr. Shortt removed one or two melanotic tumours from patients in Orissa.

Dr. D. Wilkie has recorded a case of carnification of the lung from the pressure of a melanotic cancerous liver. It occurred in a jockey, probably English, *æt.* 46. It is not said how long he had been in India.

Preparations 318 to 326 in the C. M. represent this form of cancer as occurring in man, and in the horse and cow.

Webb gives, at p. 1 of his 'Pathologia Indica,' the case of a native in whom there was melanosis of the lungs and heart. Here, as in the case of a woman whom I saw a few years ago in London, there was cough with expectoration of broken-down melanotic cancerous matter.

Colloid Cancer is represented in Ewart's Catalogue, Preparation 327. The growth affected the mesentery. Having seen a very marked case of colloid of the stomach at Guy's, I was on the look-out for this form of malignant disease in India, but never met with it in my own practice.

¹ *I. A.*, No. 15, p. 272, case by Dr. R. Brown.

² Dr. H. Baillie's case, *I. M. G.*, vol. i. p. 62; and Ewart's Catalogue, Prep. 318.

BRONCHOCELE

Is more or less prevalent chiefly along the sub-Himalayan ranges, from Assam, in the east, to Ladak, in the west. Solitary cases are not unfrequently met with in Lower Bengal. My own experience, in that part of India, confirms that of Mr. H. M. Greenhow, who wrote: 'I have not seen a case of goitre among Europeans in India.' In 1881, only one European soldier's wife was admitted for goitre in all India. In her, the disease probably originated, as it did in Anne Boleyn, at home.

In a report on the Medical Topography of Tirhoot,¹ Kenneth Mackinnon observes that 'European adults are very seldom attacked with goitre, children very frequently, but infants never.' The editor of the journal remarks in a footnote, 'a child who arrived in Calcutta about twelve years since still has it as much as ever.'

A late head of the Bengal Medical Department, Mr. John McClelland, who paid great attention to the subject of Indian goitre in its relation to locality and soil, denied² that the neighbourhood of Indian mountains is influential in the production of bronchocele. He said: 'The districts from Rungpore to Goruckpore extend along the borders of the Terai, and this last extends along the foot of the Nepaul mountains; and so, from an association of ideas, the goitre has been here generally supposed to bear some immediate local connection with the mountains. But such is not the case, for it is found in open cultivated districts of the plain sixty miles from the mountains. In other places it may extend much closer to them, but never so as to' [lead us to] 'suppose that it can be due to their influence merely as hills.'

So Kenneth Mackinnon asked: 'Why is goitre confined' [by writers] 'to mountainous valleys or plains lying in the vicinity of great mountainous chains? I make the latter observation because the disease is as common, I believe, in the villages of Tirhoot, lying hundreds of miles from the Himalaya, as in any part of the world.'

In Indian localities, where goitre is most prevalent, it affects the lower animals. Mr. McClelland noticed that, in these districts, dogs and cats are often affected with goitre. Dr. Coates says that, in the Chumparun district, goats and sheep, dogs and horses suffer from the disease.

Mr. Bramley, the first Principal of the Calcutta Medical College, wrote that, in Nepaul, 'on one occasion a goat brought forth a kid

¹ Finch's *I. M. J.*, vol. ii. p. 33.

² *Sketch of the Medical Topography, Climate, and Soil of Bengal and the N.W Provinces*, p. 116.

with a goitre as large as its head. Puppies of a month old, bred from English dogs, are very frequently affected by it, as are also lambs.¹

Writing in 1883,² Dr. Archibald Campbell said that 'congenital goitre is not known in Nepal to occur among the human race.' Mr. McClelland wrote (page 107) that there is no authentic instance of congenital goitre, *i.e.* in a child. Mr. Bramley, however, observed³ that children are sometimes born with goitre. Mr. Baillie Fraser made the same observation in his work on the Himalayas. Mr. Greenhow confirms this from his own observation. He met many persons who had suffered from the disease from their birth.

In 1867, Dr. Colles received at the Calcutta Medical College Museum a 'bronchocele from a newly born infant,' contributed by Mr. G. D. McReddie, civil surgeon, Hurdai, Oudh.⁴

With regard to goats and sheep, Dr. Campbell found, from many observations, that: '(1) Goats and sheep brought from the North-Western Provinces of India, where goitre is unknown (?) to Nepal and Tirhoot, where it is very common, produced goitred young, though they were pregnant previous to arriving in the infected districts.

'(2) A goat or sheep unaffected, and in perfect health, produces in Nepal goitred offspring.

'(3) A female parent, having the disease, scarcely ever produces other than diseased young ones.

'(4) Of twenty-three lambs and kids born with bronchocele, all died within a few minutes after birth, with the exception of a lamb in which "one gland only was enlarged;" when it was six weeks old the disease had vanished.

'(5) The tumours in the offspring of goitred parents are invariably larger than in that of healthy ones.

'(6) The death of the affected young ones does not depend on the size of the goitre.'

The vulgar say 'the creatures die because they cannot suck.' 'Possibly they could not,' remarked Dr. Campbell, 'but they are very rarely allowed to try. The common course being this—the creature is born, is feeble and *unable to raise its head*, gasps half-a-dozen times for breath, and dies.'

He found that 'the muscles arising from the sternum to be inserted into the os hyoides and thyroid cartilages are nearly obliterated by the pressure of the tumours.' A further examination of these remarkable cases in children and in the lower animals, by comparative anatomists, all the muscles of respiration being looked to, is a desideratum. Such

¹ As cited by Greenhow.

² *C. M. T.*, vol. vii. part 1, p. 1, 'Observations on the Congenital Goitre in Animals, as it occurs in Nipal.'

³ *C. M. T.*, vol. vi. p. 181.

⁴ *I. M. G.*, vol. ii. p. 244.

inquiry should also include a full investigation of the condition of the cranial bones of cretins and goitred animals.

Speaking of goitre as observed in Kemaon, Mr. McClelland thinks that the tumour does not always originate in the thyroid gland, and states that, in a third of the cases which he saw, it appeared to commence with a fulness of the base of the neck, on one or both sides over the middle of the clavicle. Thence the swellings ascend, and, in a longer or shorter time, reach the situation of the thyroid gland, when both tumours unite. I do not find that any other observer has noticed this; but Dr. Hilton Fagge held that, when the thyroid is absent in cretins, fatty tumours will almost invariably be found in the posterior triangles of the neck.

With regard to the *Prevalence* and *Localisation* of bronchocele in India, the returns for 1881 show 59 admissions for bronchocele in the sepoy force of the Bengal army, none among the native troops of the Madras and Bombay Presidencies. Among the prisoners, there were only two cases in Bengal and seven in Madras.¹

Following the Asiatic sites of bronchocele from east to west, it is noticed in the 'Madras Topographical Reports,'² that, in the highlands both of *Siam* and *Ava*, bronchocele is frequent, but the gland does not attain any great size, nor is this condition attended with cretinism.

Dr. Waring states, in his medical notes on the Burmese, that among some of the tribes of *Karens*, in the interior from Mergui, bronchocele is very common, but is confined to small communities inhabiting certain districts. Mr. John Leslie³ saw a good many cases at *Gowhattee*, in *Assam*, but in none was the enlargement of the gland very great.

In a tour which I made throughout *Assam*, in 1865, I saw nothing of goitre, except at one very small village on the plain country between Tezapore and Nowgong. I was surprised to see that nearly the whole of a large crowd of young children who ran out to look at my elephant had goitre.

Dr. Mouat says⁴ that 'in the districts about *Motiharee*, *Segowlee*, *Bethiah*, *Bhagolia*, and on to *Goruckpore*, indeed all along the line of the Terai, goitre is so prevalent that it can scarcely be an over-estimate to state that, in many localities, one individual in ten is afflicted. Dr. Coates has stated that 90 to 100 cases of goitre resorted daily to the *Chumparun* dispensary when he was civil surgeon. Writing in 1859, Dr. John Brown⁵ observed that, in the valley of *Nepaul* (where Dr. Campbell made his observations on goats and sheep, cited above) and its vicinity, goitre prevailed to a large extent. (*See Cretinism.*)

¹ A prisoner would only be admitted to hospital for goitre under exceptional circumstances.

² Tenasserim Provinces, p. 175.

³ 'A Sketch of the Medical Topography of Gowhattee,' *C. M. T.*, vol. vi. p. 56.

⁴ *I. A.*, No. viii. p. 437.

⁵ *Ibid.*, No. xi. p. 176.

Dr. D. Wright observed,¹ in the same locality, that the disease which supplies most patients to the dispensary is goitre. 'The door is, in fact, often besieged by crowds of applicants for *gullé-ka-dawa*' (medicine for the throat). 'Goitre is most common among the women, but is also pretty frequently met with in males; and, in certain districts of the valley, where it is most abundant, the very dogs are affected by it.' My friend Mr. Greenhow's observations on goitre² were confined to the Trans-Gogra portion of *Oude*, and more especially to the neighbourhood of *Secrora*. At that station, he easily brought together 300 cases; and, from his careful examination of these, he draws many inferences which will be found of great value by all who may investigate the subject of bronchocele. He notes that 'at Hissampore, a village on the Surjoo, about twelve miles distant from Secrora, dogs and other animals are affected with it.'

My friend Dr. Cayley observed³ that he had travelled through the greater part of *Ladak*, and had seen very few goitres, and those very small ones; and he saw no cretins, and hardly an idiot; although, in the lower Himalayan ranges, as at Kangra, Kullu, and about Simla, goitre is very prevalent, and cretins not uncommon.

In his valuable paper⁴ on Goitre in the Himalayas, Brigade-Surgeon William Curran notices that the existence of goitre on the higher table-lands of Central Asia was first made known by Marco Polo. Describing the 'Province of Yarcán,' he says that 'a large proportion of its inhabitants have swollen legs and *great crops at the throat*, which arises from some quality in their drinking-water.' Mr. Curran also cites Mr. Schuyler's observation that, in the city of *Khokand*, it seemed to him as if every third merchant was afflicted with goitre. He was told that the disease is very prevalent in *Kashgar* and *Yarkand*.

Acute bronchocele.—A report by Surgeon-Major A. A. Gore,⁵ shows that, in December 1868, Surgeon-Major L. Cameron described the 41st Native Infantry as suffering remarkably from acute goitre. They went from Agra to Buxa, an old Bhootan fort, in the Western Dooars, situated on a lower spur of the Himalayas, 1,800 feet above the sea-level and built on a confined plateau in a narrow valley. The geological formation was gneiss of varying composition, probably containing lime, as streams issuing from it encrusted twigs with carbonate. Their strength was 678; 21·7 per cent. of their number became affected with acute goitre. The shortest period of exposure was eight months, the longest thirty-nine months. No European adult was attacked, but some cases occurred among European and

¹ *I. M. G.*, vol. ii. p. 195.

² *I. A.*, No. xii. p. 435.

³ Notes on Ladak, *I. M. G.*, vol. iii. p. 5.

⁴ *Dublin Journal of Medical Science*, March 1866.

⁵ *I. M. G.* for December 1884.

native children. The Bhooteas, residing in the village north of the fort (284 in number) suffered thus: men, 5 per cent.; women, 7·7; children, 2·9. The Bhooteas suffered less than the regiment, less even than the Hill Hindoos. There were 16 second attacks; average interval, 10·1 months; average age, 24 years. The 38th Native Infantry succeeded this corps at Buxa; average strength 598, chiefly Hindoos. The admissions for goitre were $7\frac{1}{2}$ per cent. Dr. Courtney noticed that the total number of cases in the Bengal Native Army, from 1873 to 1875, was 260, out of an average strength of 53,350; so that one-half of the cases occurred in a well-known goitrous locality. In most cases, the disease commenced as a soft swelling of, generally, the right lobe of the gland, gradually spreading and becoming somewhat indurated. Frequency of pulse and palpitation were not observed. The treatment generally lasted from three to six weeks. Two applications of the red iodide of mercury ointment (sixteen grains to the ounce) with the administration of Lugol's tincture of iodine, were, in most cases, found sufficient.

The 30th Regiment landed in India, February 9, 1880. In 1881, they had, at Ranikhet, 13 cases of acute goitre, 1·3 per cent. being attacked. The average time in the hills of the 13 patients was 365 days, minimum 186, maximum 563. None of the regimental women or children or visitors to the station were affected. The treatment by biniodide of mercury ointment, as recommended by Dr. Mouat, was entirely successful. During 1882, there were sixteen admissions, in an average strength of 931.

Some remarks by Assistant-Apothecary W. P. Heher¹ appeared in 1881, founded on the case of a native (cooly) treated at the Mussoorie Dispensary, where it does not appear that the affection in its acute form is apt to occur. It seems to have been an old goitre, in which, within sixteen days, acute inflammation caused very considerable enlargement. He cites the account of an outbreak of acute goitre given by Aitken [vol. ii., 7th ed., page 1,008], in which, in the garrison of Briançon (Hautes Alpes), a strength of 48 officers and 954 men had 51 soldiers and two *enfants de troupe* admitted in 15 months. One case occurred eight days after arrival. 'The sanguino-lymphatic temperaments and robust constitutions were most attacked.'

Exophthalmic bronchocele.—I never saw this condition in India, but have heard of the case of a young lady, born in India, who probably had a strain of native blood, in whom it was present. I find only one case, in a soldier's wife, mentioned in the returns of the four years ending 1883.

A full report upon the morbid anatomy of Indian goitres, in man and animals, would be a very desirable addition to pathology.

A great deal has been written by McClelland,² Professor Liston,

¹ *I. M. G.* for March,

² McClelland, *op. cit.* p. 112.

Greenhow, and other Indian observers, with regard to the *geology of the localities in which goitre prevails*. The whole of the interesting results deserve to be carefully weighed by those who may desire to investigate this important subject, particularly in the localities themselves. McClelland and Liston are agreed in the following observations upon localities in the Goruckpore district : 'Goitre or *geega*, as it is called, is very prevalent where the soil is white, damp, and sandy ; such lands being called *bhat*, as not requiring irrigation for the dry weather crops. It is unknown in *bangar* lands, where the soil is reddish, sandy, and dry, so as to require irrigation in the dry weather. There is, however, no remarkable difference of level in the two kinds of land generally. All the villages on the *bhat* lands are not equally affected ; the inhabitants of some being quite exempt, while the inhabitants of others are affected to the extent of ten per cent. of their population ; women being more subject to the disease than men.' Professor Liston observed that the *bhat* is a soil retentive of moisture, and that a very notable proportion of it is calcareous matter. 'The *bangar* country,' he says, 'is more congenial to the human constitution, and to horses and dogs, than *bhat* lands are. The inhabitants are better grown, stronger, fairer, handsomer, and the children more playful in the former than in the latter.'

I read, in Mr. Pugin Thornton's article 'Goitre' in Quain's Dictionary, that 'in the Indian Punjaub, where goitre affects 60 per cent. of the population' [?], '59 grains of lime have been found in a gallon of water, 10 grains being an undesirable proportion' (Professor Frankland).

In 1872, Dr. Francis Macnamara, the chemical examiner to Government at Calcutta, commenced a series of analyses of waters obtained by the medical authorities from Indian districts in which goitre is prevalent.¹ From these examinations he concludes that 'the lime and magnesia theory of the disease is not tenable.' He further concludes that goitre is caused by malaria.

The *Treatment* of goitre by the inunction of biniodide of mercury ointment, which, I believe, originated in India, met with so much success there, and has for so long a time been noticed in the medical literature of Europe, that it appears surprising that we should have heard so much, of late years, of the very serious operation of thyroidectomy.

Writing in 1857, Dr. Mouat stated that, in 1854-55, Captain Cunningham, second in command 12th Irregular Cavalry, began to employ the biniodide ointment. I do not find it mentioned who suggested this plan of treatment to Captain Cunningham. He appears

¹ *Climate and Medical Topography in Relation to the Disease Distribution of the Himalayan and Sub-Himalayan Districts of British India, with Reasons for Assigning a Malarious Origin to Goitre and some other Diseases.*

to have been speedily satisfied of its efficacy, as we are told, in Dr. Mouat's paper cited above, that, in the first winter, he must have treated some 25,000 cases of goitre, having seen as many as 500 at his house in one day. Dr. Mouat considered that, in about three years, not less than 60,000 persons had been treated.

I am indebted to Dr. L. A. Waddell, resident physician Medical College Hospital, Calcutta, for an interesting contribution to our knowledge of the pathology and therapeutics of bronchocele.¹ Dr. Waddell, having examined the blood microscopically in four cases of goitre, found that anæmia was not present in either. In two of the cases, there was decided plethora of blood-corpuscles, and the hæmoglobin was also above the average.

In 1881, Dr. E. Noakes, who for several years had been using hydrofluoric acid in the treatment of goitre, published in the 'Lancet' ² a report giving the results of twenty cases treated by this method. He placed the recoveries at eighty-five per cent., concluding that the acid may justly be considered an anti-goitrous remedy which, although not a specific, deserves to be regarded as a basis of constitutional treatment. Reviewing Dr. Noakes's cases (which remained under treatment from 3 to 13 months, with an average of $7\frac{3}{4}$ months), Dr. Waddell infers that Dr. Noakes's percentage of recoveries requires to be considerably modified; but, then, even with this qualification, a large margin of undoubted success must still be allowed to the hydrofluoric acid treatment. In two cases, treated by Dr. Waddell, the sodium fluoride was taken with great disgust.

He considers that, in simple soft enlargements of the entire gland, by persisting with the fluorides, one may reasonably hope to effect ultimately a substantial reduction of the tumour, if not complete cure. It is clear that the agent is of but little avail in hard fibrous enlargements. It is not probable that this plan of treatment will become established in India, considering the slowness of its action when small doses are given, and the intolerable nausea produced by large doses. It is held that 'considerable anæmia,' a condition to which all in India are sadly liable, 'must attend the prolonged use of the drug, even in small doses.'

As, however, it appears that hydrofluoric acid and the alkaline fluorides exert a decided therapeutic influence upon goitre, and as it is to be conceded that their use in combination with other agents may not improbably be found advantageous, this subject merits the full attention of Indian practitioners.

The observations made by Dr. Coates in 1881, already cited, are among the latest on Indian goitre that I have seen. He said that the whole of

¹ *The Physiological and Medicinal Action of Fluoric Acid and the Fluorides*, reprinted from the *I. M. G.*

² *Lancet*, vol. i, for 1881, p. 448.

his very numerous cases at Chumparun were treated with an ointment composed of biniodide of mercury 3 ix to lbs. iij of lard. His experience was that one-third recovered completely, in one-third the size of the tumour was reduced, and in one third no change took place. He considered it of importance to expose the part to the full rays of the sun during and after the application of the ointment.

I had an opportunity of witnessing that issue which is threatened in cases of unchecked bronchocele. An old native woman was brought into my ward in a very low state. She was attacked with symptoms of suffocation and died unrelieved, as there was no room to allow us to open trachea or larynx, and as it was clear that she could not have survived an attempt to remove the tumour. I found that the air-passage was surrounded and pressed upon by the tumour as it would be if grasped in front by the palm of the hand and embraced behind by the thumb and fingers joined, save that the tumour was bulkier posteriorly.

CRETINISMUS.¹

Wherever bronchocele is extremely prevalent in India, cretinism may be found.

Our local medical literature contains a considerable amount of observation upon this disease.

Writing in 1859, Mr. McClelland stated that, in one locality alone, during a halt of a few hours, Mr. Nicholson, of Bubnowlee, in the Goruckpore district, collected numerous children—*bowkuts*, or cretins—from an adjoining village for his inspection, as examples of the condition of the people in large tracts of country in his neighbourhood. He found that ten per cent. of the children of the villagers where goitre prevails are *bowkuts*, or idiots. Many are deaf and dumb; their limbs weak and feeble; their features large and without expression; the eyes dull and vacant. They seldom live to attain adult age. Cretin children in these districts are generally, but not always, born of goitred parents. Sometimes one parent has goitre: more rarely, the parents are free from the disease. Cretinism begins to show itself at a very early age; the goitre comes on later. Both forms of the disease sometimes go together in the same individual; but there are many instances of cretins without goitre. Mr. McClelland's remarks on the association of goitre and cretinism in Kemaon² deserve close attention. He considered that these diseases are intimately connected. He observed that, in the little hamlets where goitre prevailed to a great extent, the people were characterised by a want of enterprise and bodily vigour, as compared with their immediate neighbours, who were

¹ I have thought it well to place Bronchocele and Cretinism together.

² *Op. cit.* p. 105.

free from the disease. 'In Goseragong, the people are generally affected with goitre, yet there are no cretins among them. The same may be said of Deota; but, in the villages of Salmora, Oriel, Goraght, Tomilly, and Ager, which contain 138 inhabitants, 78 had goitre, and 42 were cretins; while there was not one of the latter class to be found in any of those villages that were exempt from goitre. Hence it appears that, in a population in which goitre prevailed to the extent of rather more than 50 per cent., 30 per cent. were cretins; while, in the remaining portion of the people, amongst whom goitre did not extend to above 9 per cent., there were no cretins.'

In that variety of goitrous disease which occasions cretinism, the patient is invariably seized before the age of five years, and the disease continues uninterruptedly throughout adolescence. During this time, there is a depraved action of the absorbent system, shown in the monstrous development of certain organs; while the natural growth of others is proportionately suspended.

Hence the limbs are short and crooked, the spine distorted, the head often of enormous size, the features bulky and idiotic, and glandular swellings are common on various parts of the body, and seldom entirely absent on the neck. The cretins are not, in general, so imbecile as natural-born idiots. They retain, at least, some glimmering of reason acquired in childhood, which enables them to perform many useful offices. Cretins are sometimes seen without any great enlargement of the thyroid gland, but such cases are rare, and they are generally much deformed, so as not to be mistaken for congenital idiots. Tumours on the elbows, knees, and other joints, as well as along the course of the lymphatics, are common with the cretins of Ager and other villages in Kemaon.

In writing upon the diseases of Nepaul,¹ Dr. D. Wright notices that, in that country, cretinism is, as usual, an accompaniment of goitre, but the cases seen in Nepaul are by no means very numerous in proportion to the number of goitrous people that are met with.² Writing eleven years earlier, Dr. Wright's predecessor, Mr. John Brown, observed that 'in the Valley of Nepaul and its vicinity, goitre and, in many instances, cretinism prevail to a large extent.' He adds that the towns of Deopatun and Nunksal 'are affected with goitre and cretinism to a large extent.' In his valuable 'Observations on Goitre as seen in Oude,' my friend Mr. H. M. Greenhow³ maintains, apparently with too much confidence, that cretinism 'is quite unknown throughout the Himalayan regions, and that, according to his own experience in Oude, it is equally unknown there.' Out of his 300 cases of goitre, he only met with one individual who showed the least sign of weak intellect. This was a boy who was not deformed in body in any

¹ *I. M. G.*, August 1, 1867.

² *I. A.*, No. xi. p. 176.

³ *I. A.*, No. xii. p. 449.

way, but whose mind was evidently imbecile. He urges that it is not surprising that, in such a number of persons brought indiscriminately together, one should be found of less than ordinary capacity. Those he examined were generally as healthy and strong in mind and body as are the country people in any part of India he has visited. It will be noticed that, while stationed for a winter at Secrora, Mr. Greenhow 'sent his servants and other agents out in the bazaars of the town, to bring to him any goitred persons whom they might encounter; and, in this manner, he soon brought together 300 cases.' Probably, these were mostly sufferers of exceptional vigour anxious to obtain relief by medicine, while their torpid cretins remained inactively at home. At any rate, a special search for cretins by medical men who may travel through that district appears desirable.

Dr. Coates stated, in 1880,¹ that Chumparun is a peculiarly goitrous district. Goats and sheep, dogs and horses, suffer from the disease. Cretins (*bods*) are frequently met with in goitrous villages.

In his paper on 'Goitre,' which I have cited in my notes on that disease, in speaking of the great prevalence of bronchocele in the districts about Motiharee, Segowlee, Bethiah, and on to Goruckpore—indeed, along the whole line of the Himalayan Terai—Dr. Mouat says that, in reply to the question: 'Have you seen any cases of goitre patients whose intellect was affected by the disorder?' Captain Cunningham, who had treated some 25,000 cases of goitre, replied: 'I have seen many cases of goitre patients whose intellect was decidedly affected by the disease; as, after the goitre was reduced, they appeared much more intelligent, and like rational creatures, which they were not before.' He added: 'However, there are so many idiots about this part of the country, only slightly affected with goitre, that it would be difficult to say whether idiotcy was actually induced by it, or the symptoms increased in those whose intellect was naturally defective.'

Between Landour and the Snowy Range, Dr. J. Fleming saw many cases of goitre, but few cretins.

Surgeon-Major Ross² found goitre very prevalent in Kashmir, especially in the Sind Valley. It almost exclusively affects females. 'Some cretins are to be seen.'

In a paper on the Medical Topography of Orissa, published in No. X. of the 'Indian Annals,' Dr. Shortt makes bare mention of the fact that 'cretins are met with in many parts of the Hill Districts.'

I am not aware that the above remarks by Brown, Wright, Greenhow, and Mouat, have led to any systematic inquiry into the subject of cretinism in the Sub-Himalayan districts. I shall be grateful for further information upon this important subject from brother officers who may visit those localities. Late research having shown that cretinism may attend congenital deficiency, or acquired disease of the

¹ *I. M. G.* for that year.

² *I. M. G.* for October 1878.

thyroid, and may follow its ablation, it will be well to obtain additional observations upon the mental condition of those innumerable native patients in whom goitre has disappeared under the use of biniodide of mercury ointment. In conducting such inquiry, valuable data may be looked for in the observation by Professor Kocher and Dr. Lardy, of Berne¹ who declare that a progressive cachexia, designated by Kocher *cachexia strumi priva*, is very liable to be induced by extirpation of bronchocele. This they find to be characterised by great anæmia, tumefaction (especially of the face), and a diminution of intellectual activity, furnishing a display of symptoms resembling those of cretinism. Dr. Lardy adds that a remarkable feature in these cases is 'the brutalised countenance these unfortunate persons assume, while they become incapable of executing any employment that demands intelligence.' We are told that Professor Billroth, who has himself performed thyroidectomy 120 times, entirely denies that this condition is liable to follow the operation.

The experience of MM. Jaques Louis and Auguste Revertin² confirms the observation of MM. Kocher and Lardy. They noticed torpor of intellect, puffiness of the face and extremities, which, however, did not pit on pressure, and an alteration in the complexion, a whitish yellow colour, not that of anæmia or albuminuria, but similar to what is seen in some cretins. It is added: 'These symptoms closely resemble some of those noticed in cases of *myxœdema* which have been recorded by Ord, Hadden, and others, and the similarity is especially noteworthy from the atrophy of the thyroid gland which has been observed.' A very important discussion on the association of cretinism and myxœdema with changes in the thyroid gland took place at the Pathological Society on November 23, 1883, and it was understood that the society would appoint a commission for the investigation of this question.

Although bronchocele is not endemic in the Delta of the Ganges, many cases present themselves there. I did not see cretinism or myxœdema in Lower Bengal.³

DIABETES.

Glycosuria is frequent among rich elderly natives in Calcutta, many of whom are fat and indolent and immoderately fond of sweet-

¹ Beilage zum *Centralblatt für Chirurgie*, and *Union Médicale*, December 6, 1883. *M. T.*, vol. ii. for 1883, pp. 273 and 693.

² Review of their note: 'Sur vingt-deux Opérations de Goitre,' *M. T.*, vol. ii. for 1883, p. 527.

³ For numerous data relative to bronchocele in India, see an article from the *British and Foreign Medical Review*, fully cited in the *I. M. J.*, vol. v. p. 47, N. S., for 1840 and an article on 'Bronchocele,' by Mr. Brett, *Ibid.* vol. iv. p. 438, N. S.

meats. I heard of several fatal cases of carbuncle in such diabetic patients.¹

Writing in 1871,² the Editor of the 'Indian Medical Gazette' remarked that he believed that 'among the upper and middle classes of natives in Calcutta, almost every family had lost one or more of its members from diabetes.' Baboo Satcory Dutt had informed him that, in one of the most respectable and influential native families in Calcutta, No. 1 died of acute diabetes at the age of 65; No. 2, his eldest son, of chronic diabetes; No. 3, his third son, had chronic diabetes; No. 4, his fourth son, died of acute diabetes; No. 5, daughter of No. 1, now aged 65, has diabetes; No. 6, eldest son of No. 2, grandson of No. 1, died of chronic diabetes; No. 7, second son of No. 2, has diabetes; No. 8, eldest son of No. 4, has diabetes. In addition, the wife of No. 3 died of diabetes.

I saw, in India, three European gentlemen suffering from this disease. These cases were very chronic. Two of their number attained advanced ages. The third, a Bengal civilian, was found to be diabetic, by one of our best authorities, about sixteen years ago. As he is now a very robust man in green old age, it appears likely that the glycosuria was due to a transient cause. This case is probably explained by Dr. Wilks's observation that, in young diabetic patients, the tendency is sooner or later towards a fatal termination; whilst, in the old, the presence of sugar in the urine is often found in those who are suffering from mere dyspepsia or gout. Cases of diabetes have frequently been detected in India by noticing that flies and trains of large black ants visit the utensil. Morehead gives an account of six cases of saccharine diabetes in natives, all the instances of that disease of which he had personal knowledge in India. (For an allusion to *Pancreatic Diabetes*, see Diseases of the Pancreas.)

Dr. Cayley relates³ a case of removal of a scrotal tumour in a native which terminated successfully, although diabetes appeared three weeks after the operation, and recovery was retarded by repeated attacks of secondary hæmorrhage. In 1874, when the milk system in glycosuria was much under discussion, I showed, in the 'Indian Medical Gazette,' that, in his 'Phthisiologia, or a Treatise of Consumptions,' Dr. Richard Morton, who died in 1698, gave histories of milk treatment in diabetes.

PURPURA.

I did not see *Purpura Hæmorrhagica* in India, and I have met with detailed records of but very few cases.

¹ Baboo Kamikha Nath Acherjee gives, in vol. ii. p. 201 of the *I. M. G.*, two cases of carbuncle and boils in well-to-do natives in which the characters of these diseases led him to discover that diabetes was present.

² *I. M. G.* for December, p. 259.

³ *I. M. G.* for August 1875.

Small, but fatal, outbreaks of *Purpura* occurred in the four years ending 1883. In 1880, 4 European soldiers, 1 death, 1 fatal case in a child; 4 sepoy admitted, with 2 deaths. In 1881, 4 European soldiers admitted; 3 children, of whom 1 died; 1 prisoner, who died. In 1882, European soldiers, 3 admissions, 1 death. In 1883, European soldiers, 7, 1 fatal; 1 child admitted, and a sepoy who died.

In a report upon the health of H.M.'s 54th Regiment in the Madras Presidency, Mr. Hamilton noticed ¹ that six cases of purpura hæmorrhagica had been under treatment, one proved fatal; the immediate cause of death was effusion into the thorax after a very tedious illness. Two men were invalided for this disease. In the treatment, more decided benefit was derived from the employment of quinine, with liberal diet and moderate use of wine, than from any other means.

There is ² a very full report, by Dr. Gamble, of a case of purpura occurring in a private of the K. O. R. at Bellary, Madras Presidency. There were considerable ecchymosed spots on some, and vibices on other parts of the body. There was also hæmoptysis. Two ounces of pure blood of arterial hue were spit up daily. There appear not to have been any febrile symptoms. On the fourth and seventh days, there was profuse epistaxis. On the twelfth, there was considerable hæmatemesis, followed by melæna. Oranges were then allowed. Slight epistaxis recurred from time to time, but he was convalescent in less than three weeks. According to the practice of the time, V.S. to eight ounces was employed on the twelfth day after the 'considerable hæmatemesis.' We are told that 'the blood drawn separated after a few hours almost entirely into serum; the crassamentum consisting of a mere scum, occupying the whole surface of the bleeding-cup, of a dark sooty colour.'

Mr. Raleigh ³ treated, in 1834, a stout well-made sailor, aged 21, who, having come from America in 123 days, had been about two months in Calcutta. On the voyage, he had been fed principally on salt provisions; but, since coming into port, had been liberally supplied with fresh meat and vegetables. He had invariably been in good health at sea, and, during his last voyage, 'had been perfectly free from scurvy' or other complaint. When he had been about a fortnight in port, a 'red rash' appeared, without any accompanying indisposition, on his face, chest, arms, and thighs. The spots became daily more numerous, larger and darker, for six weeks. Three days before his admission, 'copious bleeding took place from the mouth, from which a constant draining of blood had continued.' The reporter does not precisely indicate the source from which he considered that this hæmorrhage (the stools frequently contained large quantities of blood) proceeded, but perhaps the explanation appears in the following paragraph:

¹ *Madras Medical Quarterly Journal*, vol. iv. p. 329.

² *Ibid.* p. 369.

³ *I. M. J.*, vol. iii., N. S., p. 734.

‘On the dorsum of the tongue, were five black *elevated* spots of the size of a pea, and, on the inside of each cheek, were two others of the size of a sixpence with irregular edges, the soft palate and uvula were thickly speckled with petechiæ, the gums were *not* ulcerated.’ He had no contraction or stiffness about the joints. The face, chest, and trunk generally, but particularly at the sides, over the shoulders and nates, were thickly covered with spots, varying in size from that of a pin’s head to the circumference of a large pea, and of various shades of colour, between bright purple and dark brown, not elevated. On the forearms and nates, were several large vibices in stripes, as if produced by the lash of a large whip; these were very black, and irregularly raised so as to feel rough. His countenance was rather sallow. He declared, on admission, that he experienced no uncomfortable sensations or pain. His appetite was good, and he slept well. In the first ten days of his treatment, there was occasional mention of feverishness and headache. There was considerable bleeding from the mouth for four days in hospital. From the fourth to the sixth day, much blood passed by the urine, which, for the first two of these days, was loaded with mucus. Upon the principle which then ruled practice—that hæmorrhage indicates V.S.—he was bled eight times from the arm during the first nine days ‘and lost at least 120 ounces of blood, independent of the enormous quantity which flowed from the mouth and was voided by the stools and urine. He took a powerful cathartic daily.’ On the sixteenth day after admission, he was reported ‘perfectly recovered, a little weak.’ It is added: ‘He was never so much reduced by the treatment as to be confined entirely to his bed. I found him daily walking about the hospital, and he always walked to the door or window of a morning, in order that I should examine the eruption.’

My friend, Dr. Herbert Baillie, recorded¹ the case of a male infant to which he was called when it was nearly exhausted by hæmorrhage from the gums, which he had lanced five days previously. The bleeding was with difficulty stopped by the application of nitrate of silver. There was the peculiar flea-bite appearance seen in purpura over the body and limbs. Dr. Baillie then recalled the fact that, during the two preceding months, there had been an occasional appearance of maculæ on the trunk. There would appear a little lump under the skin, nowise discoloured. In two or three days, the swelling increased and was accompanied by an erythematous blush, which, in a few days more, extended, and became of a deeper hue, leading to the belief that supuration was about to take place, when, in another day or two, being then larger in circumference than a rupee, the place became bluish, then green, and disappeared gradually like a bruise. The decline of these patches closely resembled the changes seen in cases of erythema

¹ *I. A.*, No. 7, p. 297.

nodosum, which it also resembled in the presence of fever. The house being low and damp, the family removed the child to a boat on the river, and to Calcutta, where recovery was speedy.

In 1878, Mr. Harden reported a fatal case of purpura hæmorrhagica in a Hindoo boy of 10. There was dry gangrene of both feet.¹

TRUE LEPROSY.

Such full accounts of this disease, as it occurs in India, have been given in works accessible to all English readers,² that I shall now do little more than state a few results of my own observation. I constantly saw cases of this disease throughout my long Indian service, and I was officially desired to make trial of (1) the cashew oil and (2) the gurjon oil upon cases in the Medical College Hospital.

The disease presents itself in natives of all ranks, seldom in the rich and well-to-do; but I have seen it in a sovereign prince and in a rich man. It is scarcely proved that it can attack Europeans. I saw, only for a moment, a boy of European appearance, in whom I considered that the disease was commencing; it was said that, many years ago, a Madras Medical Officer was leprous, and there was strong suspicion of another case; but, in India, many of the fairest 'European' aristocrats are descended from native ancestors, and leprosy is not rare among half-castes.

The census report of the North-Western Provinces for 1881 gives nearly 18,000 lepers in a population of nearly 33,000,000.³ Mr. Peters makes, upon the authority of Mr. McCorkell, C.S., a statement, from the census of 1872, showing that, in the Collectorate of Belgaum, in Western India, the number of persons disabled by leprosy was 4,131 in a population of 938,750, or 0·44 per cent. Yet, in the Belgaum Leper Asylum, and from its neighbourhood, Mr. Peters could only find 29 lepers to treat in 28 months. Mr. Vincent Richards states that the proportion of lepers in Balasore is said to be about 1

¹ *I. M. G.* for March 1878.

² Among many others—Horace Hayman Wilson, on 'Kushta,' or 'Leprosy,' as known to the Hindus; *C. M. T.*, vol. i. p. 1; F. Brett, *I. M. J.*, vol. v. p. 96, N. S.; Erasmus Wilson, *Lancet*, April 1856; and Quain's Dictionary; Morehead, *op. cit.* 695; Carter, *Transactions of the Medical and Physical Society of Bombay*, No. 8, p. 5, N. S.; 'Report of the College of Physicians,' commented upon, from Indian experience, by N. C. Macnamara, in the *I. M. G.*, 1866—a highly valuable monograph, afterwards published in a separate form by Messrs. Wyman & Co., of Calcutta; 'Anæsthetic Leprosy,' by J. N., *I. M. G.*, vol. ii. p. 215 *et seq.*; Vincent Richards, *I. A.*, No. xxxi. 1873, p. 303; Dr. W. Monro, *Edinburgh Medical Journal*, September 1876 to November 1879; Drs. Lewis and Cunningham, in the 'Twelfth Report of the Sanitary Commissioner with the Government of India;' Mr. Peters, *Edinburgh Medical Journal* for March 1883.

³ *M. T.*, January 1883.

in 5,000; in the Sonthal Pergunnahs, 1 in 700; in Bancoorah, 1 in 350; in Beerbloom, 1 in 250; and in Burdwan, 1 in 450.

The latest Bengal census returns give 8·15 per 10,000, as the proportion of cases of leprosy in that province.

The statistics given in these reports cannot be received with implicit confidence, as it is not to be supposed that returning officers can always differentiate cases. Clearly, the prevalence of leprosy varies greatly in different localities. It is probably at least as prevalent in India as Sir J. Simpson has shown it to have been in the United Kingdom almost from the time of the Norman Conquest up to the reign of James I.

The fact that most Indian leper asylums have few inmates is probably due to the almost painless character of the disease, even when it is causing the most ghastly mutilations, and, consequently, to its popularity as a qualification for professional beggary. I have not seen a very great deal of it in native bazaars; but hosts of mendicants do not seek relief in the streets and by the road-sides, preferring to live upon food and alms bestowed by the wealthy with a large, if indiscriminating, bounty for which we have not always given the people of India sufficient credit.

The *Differentiation* of leprosy, especially of the anæsthetic and leucopathic form, from simple leucoderma often becomes a question of more than medical importance in India. A Hindu leper cannot inherit the estate of his next of kin, because, as one impure by reason of disease, he is debarred the sacred duty of firing the funereal pyre. The law does not compel a Hindu wife to live with her leperous husband. Consequently, European physicians are consulted in questionable cases; here, an incorrect opinion would be something worse than a clinical error. Having gained an idea of the physiognomy of leprosy from engravings and wax models, the young medical officer should, on landing in India, lose no time in visiting the leper asylum which is maintained at each of the Presidency towns—Calcutta, Madras, and Bombay. There, in an hour, he will master all that is best worth knowing with regard to the aspect and condition of the leperous.

It is especially useful to distinguish scars and simple leucoderma from the white spots of leprosy. In the dark races of India, the scar of a blister or a burn long remains white until, in course of time, pigmentation goes on so far as to leave the seat of injury darker than the surrounding skin. An old Hindu writer of mine brought me his son, a young lad, who stretched out a very suspicious-looking claw, upon which were large white scars. The father hastened to say that these were burns. Truly, but the cicatrices were anæsthetic. The leperous hand, wanting sensibility, had been used too freely in meddling with fire, and had been severely burnt. Surgeon-Major Peters notices this liability of the extremities of the leperous to burning while cooking, and also to be gnawn by rats at night.

Dr. William Palmer justly observes¹ that cases of leucoderma are more frequently brought to notice in India than in London; first, because they attract more attention by contrast with the darker skin of the natives; and, secondly, because there is an uncomfortable feeling that the white patches may be mistaken for leper-spots; but whether they are really of more frequent occurrence is, he considers, doubtful.² In examining cases, those of simple leucoderma may be at once set aside. There is no anæsthesia or hyperæsthesia of the white spots when we test their sensibility with a skewer-like point of wood,³ and no second sign of leprosy, no elevation of the white patch, no enlargement of nerves, no leonine visage or conjunctival deposit or thickening of ear-lobes or of other integument, no claw-like contracted appearance of the hands nor loss of phalanges, no scleroderma, open, deeply-excavated ulcers or suspicious cicatrices. As Dr. Palmer points out, in leucoderma, both the epidermis and the true skin, with its glands, remain normal in every respect except colour. Dr. Palmer holds that, the anæsthetic being the early form of the disease, the very mildest cases of circumscribed anæsthesia of skin are to be regarded as examples of leprosy, although these milder cases often show but little tendency to assume the more severe form.⁴

When I first went to Calcutta, I saw a case in which my friend the late Dr. Allan Webb gave me some excellent hints on the diagnosis of obscure leprosy. A pilot in the decline of life appears to have become slightly disordered in mind, having sent an insolent message to his superior. I was upon a committee who had to examine him and to report upon his condition. His appearance was European, but we understood that he had a strain of dark blood. His right forearm was considerably wasted, and the fingers, especially the little and ring finger, much contracted and bunched, and hardened like birds' claws. His countenance appeared quite natural. Dr. Webb showed us that

¹ *Rough Notes on the Common Forms of Skin Diseases met with in Calcutta*, p. 22.

² See Dr. A. Wood's case of 'Leucoderma in a Pathan,' *I. M. G.*, vol. vi. p. 178.

³ In our own country, as late as the seventeenth century, a discerning public appear to have drawn a very indistinct line between those two ostracised wretches the witch and the leper. In Scotland, witch-finders were designated as 'common prickers,' and received a fee for each witch they discovered. At the trial of Janet Preston, in 1646, the magistrates of Dalkeith 'caused John Kincaid, of Tranent, the common pricker, to exercise his craft upon her. He found two marks of the devil's making; for she could not feel the pin when it was put into either of the said marks, nor did the marks bleed when the pin was taken out again. When she was asked where she thought the pins were put into her, she pointed to a part of her body distant from the real place.'—Charles Mackay's *Memoirs of Popular Delusions*.

⁴ Dr. I. Newton has published, *I. M. G.* for 1868, p. 251, some careful observations on the microscopical conditions of the structures affected in leprosy. He found that 'leprosy is essentially an affection of the gelatine-yielding tissues.'

the ulnar nerve, on the diseased side, was thickened and nodular from the elbow downwards, and that *the ear-lobes were much enlarged, pendulous, hardened, and thickened*. Dr. Webb said that this condition of the ears is a crucial test in all cases of chronic leprosy, however obscure the other symptoms may be. I think that it is M. Marx who says that the French *cagots*, who are descended from lepers, are distinguished by not having the ear-lobes developed. Soon after I read this, I noticed that one of the ear-lobes of a handsome English lady was entirely wanting, evidently by congenital defect. In my experience, Webb's test has never failed, but the deposit may be slight. 'Rotundity of the pulps of the ears' is one of the signs of leprosy given by Barnhard Gordon, in his early description of English leprosy, cited by Sir J. Simpson.

In June 1883, Dr. Thin stated, at the Medico-Chirurgical Society,¹ that a bacillus has been found in Norwegian leprosy by Hansen, in the leprosy of Southern Europe by Cornil and Suchard, by Majocchi and Pellizari, and in a leper from South America by Kœbner. Dr. Thin found it in three cases of Chinese leprosy, in material supplied him by Dr. Manson, of Amoy; in West Indian leprosy, in tubercles sent him by Dr. Hillis, of Demerara, and in the epiglottis of a leper patient who died in New South Wales. He also observed bacilli in the blood of a leper patient from Hindustan.

Popular opinion, in Europe and in India, has generally held leprosy to be *contagious*; but I am unacquainted with any indisputable facts in support of this idea. I knew a Calcutta leper, of European habits, who had been married for many years to a woman who appeared to be perfectly free from the disease. Mr. Vincent Richards mentions the case of a Mohammedan shopkeeper 70 years old, married fifty years, with two children, whose wife was not affected.

With regard to *cause*, heredity has been frequently traced. In analysing 191 cases observed in Balasore, Mr. Richards found that, in over 44 per cent., the father was affected; in only 30 the mother; and, in 13 per cent., both the father and mother: 53·40 per cent. had relatives affected by the disease. Bad food, damaged grain, and putrid fish, in insufficient quantity—to use Mr. Vincent Richards's words, 'any diet deficient or redundant in particular elements'—are doubtless operative; but no medical man of Indian experience will agree with those who maintain that the disease is mainly due to a fish diet. Leprosy is rife in districts where fish is scarcely known; and, as Mr. Peters shows, natives whom caste prejudices deprive entirely of the use of fish are victims of this disease.

Hoarseness, evidently due to laryngeal disease, has frequently been noticed in Indian lepers. In England, a writ *de leproso amovendo* (Fitzherbert, Not. Brev. pp. 520-21) was made applicable to those

¹ M. T., vol. i. for 1883, p. 713.

lepers who appeared in the sight of all men that they were lepers 'by their voice, and their sores, the putrefaction of their flesh and the smell of them.' Barnhard Gordon mentions, among the infallible signs of English mediæval leprosy, 'voice nasal,' in the early stage of the disease; and, later on, 'voice hard and barking.'

Dr. James R. Jackson¹ relates the case of a leperous woman in whom ulceration, partly destroying the nasal and palate bones and removing the uvula, extended to the rima and larynx, producing œdema glottidis and necrosis of the thyroid cartilage. Laryngotomy gave relief, but she died in about a month from extension of the laryngeal mischief, and 'rapid effusion into the lungs.' She inherited the disease from her mother. Her brother was similarly diseased.

Until we become accustomed to treat them, we are surprised to find how readily leperous sores often heal. In 1876, Dr. Cayley successfully extracted an opaque lens in a leper.² Mr. Birch amputated the great toe of a leper, aged 35, and the foot of another, aged 22; both made good recoveries.

With regard to *Treatment*, I have no faith whatever in the *specific* efficacy of any lauded remedy—chaulmogra oil, the cashew, the gurjon oil treatment, in fact of any plan that had its trial and its day while I served in India.³ Hitherto it has, I believe, been a law, subject to no exception whatever, that, treat him and improve his condition to the utmost, *he who is once a confirmed leper continues to be a leper until he dies*. Some years ago, the late Dr. Bhau Dhajee, of Bombay, an able physician of great experience in leprosy, brought out an atlas of very large and beautiful photographs displaying the appearance of lepers before and after treatment. To one unacquainted with this disease, nothing could well be more complete and apparently radical than the appearance of cure in most of these countenances; but one to whom the leper visage is familiar sees, in these, as in many other cases of 'recovery' which I have examined, that, whether the face of the leper be emaciated or fat, ugly, menacing and rugged, or smooth, pleasant and comely, it still bears the ineradicable stamp of leprosy. The fact is that, in a large proportion even of fully developed and advanced cases, cachexia may be so far removed, mainly by good feeding, as to give the patient a fat and healthy appearance. Under treatment, say by the gurjon, cashew, or chaulmogra oils, tonics, and good food, the ulcers often heal readily, and the patient's general condition is vastly improved. He was a cachectic leper, he becomes a

¹ *I. M. G.*, November 1, 1866, p. 339.

² *I. M. G.*, April 1876.

³ I have no experience of Dr. J. M. Fleming's mode of 'curing' leprosy by carbolic acid, *I. M. G.*, vol. vi. p. 14; or of Mr. Lawrie's practice of nerve-stretching in anæsthetic leprosy. *Ibid.*, September 1878.

robust leper ; but he remains as essentially a leper by constitution as he ever was ; and, his good food, oleaginous frictions, and other comforts being taken away, he will assuredly fall back into a state of unmistakable leprosy. Consequently, we cannot cure leprosy—we cannot touch the constitutional disease therapeutically—but we can make the leper fat and comfortable, and thus, doubtless, greatly prolong his life.

Unquestionably a generous diet and the free inunction of *ghee* (clarified butter) and oil, after the native manner, are among the leading indications in the treatment of leprosy. A poor dietary, such as is allowed in certain Indian asylums, is a great mistake and failure of charity. A half-starved, voracious dyspeptic, such as the leper generally is when we begin to treat him, requires the best food and, as his digestion improves, plenty of it. The management of English lepers in the middle ages appears to have been simple. They were strictly confined, in habits of religion, to lazaret-houses, where they had a singularly liberal and varied diet.¹

Dr. Crombie has made some interesting observations² on *Ainhum*, or spontaneous amputation of the fingers and toes, as occurring in Eastern Bengal.

He refers to the publication of Drs. Tilbury Fox and Farquhar, p. 20. Dr. Wise gave him his first information of two cases which occurred in his practice, and of which he made an official report in January 1873.

Quoting Dr. da Silva Lima, of Bahia, Dr. Sangster states, in Quain's Dictionary, that *ainhum* 'is peculiar to the African race,' being found not only amongst the inhabitants of the West Coast of Africa, but also amongst the Hindoos of African descent, as well as amongst the slave population of South America. Assuredly there can be very little suspicion of an admixture of African blood among the Caucasian natives of Eastern Bengal.

¹ Surtees mentions that, at Sherburn Hospital, 'the daily allowance of the lepers was a loaf weighing five marks, and a gallon of ale to each ; and, betwixt every two, one mess or commons of flesh three days in the week, and of fish, cheese, or butter, on the remaining four. On high festivals, a double mess ; on the feast of St. Cuthbert, in Lent, fresh salmon, if it could be had ; if not, other fresh fish ; and, on Michaelmas-day, four messed on a goose. With fresh fish, flesh, or eggs, a measure of salt was delivered, the twentieth part of a razer ; when fresh fish could not be had, red herrings were served, three to a single mess, or cheese or butter by weight, or three eggs. During Lent each had a razer of wheat, to make furmenty (*sinulum*), and two razers of beans to boil. Sometimes greens or onions, and every day, except Sunday, the seventh part of a razer of bean meal ; but, on Sunday, a measure and a half of pulse to make gruel. Red herrings were prohibited from Pentecost to Michaelmas ; and, at the latter, each received two razers of apples.'

² *I M. G.* for August 1873, and June 1874.

Dr. Colles found these appearances in a finger which had separated. The specimen is in the Medical College Museum :—‘Distal phalanx normal in shape; its cancellous structure opened out, and filled with yellowish oily fat (commencing fatty degeneration); distal inter-phalangeal joint intact; distal end of second phalanx bony, the remainder of the bones converted into fibrous tissue; no bone at point of spontaneous amputation; fatty and fibrous tissues increased in quantity; skin moderately hypertrophic.’

In a case in which a little toe had been removed, Dr. G. C. Roy found that ‘the place of the first phalanx was supplanted by an indurated tissue surmounted by the shell of its cartilaginous articulating surface; so that, during amputation, the knife passed smoothly through its structure. Under the microscope, it was found to consist of white fibrous tissue, in which were sparsely interspersed fusiform nucleated cells and oil-globules.’

This disease is not absolutely confined to Eastern Bengal. It has been observed by Mr. C. J. H. Warden at Bhaugulpore in a native cultivator who had always resided in that district.

Ainhum appears to be a form of leprosy corresponding with the ‘Joint Evil,’ or ‘Coco Bay,’ of the West Indies. Richard Towne clearly describes the joint evil as leprosy in his work on the ‘Diseases of the West Indies,’ published in 1726. He gives the following description of the local mischief: ‘The nails curl inwards, and the extremities of the fingers and toes begin to ulcerate. These ulcers, which never digest, but generally look dry, without much foulness or fœtor, gradually creep from joint to joint, till they have eroded,’ i.e. eaten away, ‘all the fingers and toes.’

In India, this fatty degeneration of bone and other structures also occurs in larger joints, as in the ankles and wrists. This subject will be illustrated under the heading ‘Fatty Degeneration of Bone,’ in the chapter on Diseases of the Bones.

Struma.—I have thought it best to consider this constitutional condition, as it is observed in India, under the heading of *Phthisis*.

RICKETS.

I think that, at present, the occurrence of *Rickets* among natives and Europeans born in India is not proved. Several of my brother officers, of great experience in the Upper Provinces, tell me that they have never seen it. Just as this sheet was going to press, I put this question to my friend Surgeon-General W. Burns Beatson, now of Bath. He said emphatically: ‘I never saw rickets in India. Had I not known the disease when I went there, I should have been obliged to learn what it is on my return!’ Preparations 1,181, 1,249, and 1,250 in Ewart’s Catalogue are specimens of bones deformed by

rickets, but they are not stated to have been from native subjects. This is also the case with many specimens in the Grant Medical College Museum, Bombay. I do not find that any Indian observer has written on the occurrence of rickets in the country. Certainly the disease is not common in Indian hospitals. During my long experience as a clinical physician, I never said to my students, 'Here is a typical case of rickets.' But the cause of this rarity needs investigation. Thus—(1) Struma and phtthisis occur among the natives of Bengal, but far less frequently than among the inhabitants of London. Does rickets follow a similar law? (2) Did not rickets frequently occur before Glisson wrote upon it late in the seventeenth century? Was not Homer's model for the picture of Thersites a victim of rickets? Was not Richard III., with his crooked back, unilateral atrophy, and overhanging brow?

Did not this disease come to light only when national prosperity and the advance of medical science began to enable the puny and deformed children of the poor to live on, as no such children did in Sparta or in India when infanticide was lawful? What, in the middle ages, were 'changelings,' believed to be substituted by elves? The fact that extremely poor, semi-barbarous people, untaught in the means of securing the survival of the least fit, are not found to have rickety children in their huts is not proof that such children are not born to them. I have seen, at Delhi, a burial-ground filled with hundreds of little white tombs placed over the infants of the Great Moguls. It is rather the exception than the rule that one of the polygamous rulers of India leaves an heir of his body. Hence the law of adoption. What do all these invaluable little lives succumb to?

Many observers have noticed the rarity of *Congenital Deformity* in India; but this observation demands qualification. True, there are not many hunchbacked, bow-legged, or knock-kneed natives; but, in India, there is no scarcity, *first*, of those congenital deformities which do not impair bodily vigour. I could have collected scores of such cases, among others, that of a good-looking servant of my own, endowed with six toes on each foot and six digits on each hand, who took away a cartload of my best property. I regret that his extremities have not found place in our College Museum. Or, *second*, of those defects which qualify for a life of beggary, or which mark their possessor as lucky.¹

¹ In the chapter on 'Congenital Imperforation of the Lower Bowel,' I have noted that a child born without an anus is designated by the up-country natives a 'pargumber,' or 'messenger from God.' Mr. W. Cockburn states, in 'Punjaub Notes and Queries,' vol. i. p. 64, that people born with deformities, such as double thumbs, &c., are accounted very lucky, being furnished with a natural preventative against *nazar*, the evil eye. For notices of congenital malformations in natives of India, see chapters on Diseases of the Brain, Eye, Heart, Lung, Bowels, Kidney, Urinary Bladder, Female Genitals, and Pregnant State.

Vide Dr. A. Garden's elaborate report upon nine cases of malformation of the hands and feet in natives, which he observed in six years.¹ There are not many known congenital malformations which have not been seen in natives, as these pages will show.

I trust that the discussion which took place at the Pathological Society in 1880-81 has led medical men in India to take up rickets as a special study. I used occasionally to see, in the native bazaars, an object who was probably rickety. Search should be made among those professional mendicants who are cripples, always remembering that in India, as in this country, children are often crippled for begging purposes. Instances of pelvic deformity in native women are extremely rare; but a small collection of Indian cases of Cæsarian section which I have made gives countenance to the opinion that females in that country are not wholly exempt from rickety distortion impeding labour.

DISEASES OF THE NERVOUS SYSTEM.

Indian Neuroses.

As it has been shown by Fayer that nearly every one of the thanatophidia has its innoxious simulacrum, so most of the deadliest organic maladies are counterfeited by functional neuroses which, however severe and hard to differentiate, rarely kill. Thus it is with cerebral tumour, epilepsy, chorea, convulsions, angina pectoris, cardiac apnoea, croup, pyloric cancer, peritonitis, morbus Brightii, and many others.

Among the principal causes to which a tendency to various forms of neurotic suffering may be traced, are—inheritance of the neurotic and hysteric temperaments, strumous and gouty habit, the congestive action of cold, overwork of mind or body, sexual excess, tobacco-smoking, opium-eating, drunkenness, gluttony, poor feeding (scorbutus), unduly prolonged lactation, menorrhagia, leucorrhœa, bleeding piles and various other over-excitant and depressant influences, which prevail nearly everywhere. In India, we have, superadded to these, a climate which, if it fails to exhaust innervation by excessive moist and dry heat, does not recruit and give it tone by cold; and the presence, in a degree of concentration which it rarely attains in Europe, of the paludal and jungle poison. Children born in India of European parents, East Indians, and Europeans, weakened by Indian heat and malaria, are especially neurotic. Consequently, India is a good field for the study of most of the neuroses, and especially of those which simulate the organic diseases of the nervous system.

In India, the most prevalent diseases of the nervous system nearly assume the European type; but local influences, especially heat and malaria, have considerable power in modifying them. It is certain

¹ *I. M. G.*, May 1875.

that one who has an unhealthy brain can scarcely pass through an Indian hot season with impunity.

I think that it was Dr. Geddes who first showed that many European soldiers (who probably drank hard) were epileptic in India, but not in England. Extreme heat, especially when recklessly encountered by free livers, is likely to occasion every degree of mental aberration from irritability of temper to acute mania. Formerly, European soldiers used to spend many hours of the hottest days lying on their beds. A man, in the Fort of Attock, was so exasperated at a punkah cooly, who did not attend to his call 'Pull!' that he took a gun from the stand and shot him dead. Lately, one of my family was reading aloud the diary of a lady who resided in Calcutta towards the end of last century. There was an account of a man who had suddenly displayed certain evidences of mental aberration. I immediately inquired, 'In what month did that happen?' 'May,' was the reply—'Yes, just at the time of greatest heat, when weak brains are most likely to go wrong.'

Nearly the same effects upon the nervous system, as produced by extreme heat and again by unusual cold, are observable in England. Here, in the sun-stroke days which occur in almost every year, all the symptoms produced by Indian heat, from crossness, headache, &c., up to acute mania and sun-stroke, may occur, save that the Englishman, when at home, generally takes a strong nervous system into his short hot season. Still it would be by no means useless to inquire, when we hear of rash acts, 'At what season of the year did this happen?' I recollect an Indian case of the kind in which my verdict was, 'He would have acted otherwise had it been in cool weather.' The story of the old Indian Member of Council who, in the troublous discussions which used to occur in the days of Warren Hastings and Philip Francis, always had a native servant with a *lotah* of water stationed behind him with instructions to pour it over his head should his opponent's argument cause him to flush, probably has truth in it. English cold may produce any amount of brain disturbance from heaviness and headache to cerebral congestion and even apoplexy.

The Indian hot season brings out, temporarily, many little traits of defective cerebration and innervation which pass off in the rains, on return to Europe, or under the steady use of cold affusion—such as the omission or transposition of words or letters in speaking and writing, facial twitchings, nictitation, *muscæ volitantes*, iridescent lines zig-zagging about, fortification-like, beside the outer canthus, uneasy sensations down the inside of the left arm, finger-tinglings, headache, vertigo, tinnitus aurium, slight symptoms of locomotor ataxy, difficulty in rising from a sitting posture, &c.

An originally clever man, towards the decline of life, will be in a marked degree intellectually 'wanting' in India, and may greatly

recover mental tone and ability on his return to England. Eurasians generally have weak and excitable nervous systems. Men and women are prone to neuroses, and one sex is almost as liable as the other to hysteria, which, in women, is often most protean and, in many cases, goes on to mania. Among these people, we not unfrequently see forms of head and casts of countenance which used to recall an expression of Thomas Addison's. He would place the finger-tips of one hand on the patient's head, which he moved to and fro before us on its axis, fixing his eyes upon him, frowning and pushing out his lips depreciatingly, and saying, 'He has a very cerebral aspect!'—by which we understood, 'His look and the shape of his head betray a tendency to cerebral disease.'

For several years, I was a member of a standing committee to examine pilots when sick or about to invalid. The navigation at the mouth of the Hooghly is difficult and dangerous, and a very heavy responsibility is placed upon these men. The temptation to drink was considerable, but many in the prime of life, who were believed to be temperate, broke down absolutely, being quite unnerved, and declaring that they were absolutely unequal to take up heavy ships. One hot morning, an aged administrative medical officer of high rank sat opposite to me in my office, rubbing the ulnar surfaces of his forearm and hand in such a manner as to show that they were numbed. He said that it was of no consequence, he had taken strong tea at breakfast. He lived for many years after his return to England. On another very hot day, being in a Calcutta library, I was asked to see a wealthy middle-aged Armenian gentleman who had fallen. I attended to him, and drove him to his house. He had merely fainted, but, as he was recovering, there was distinct hemiplegia, which passed off at once. I knew him, apparently in perfectly good health, for many years afterwards. Sir Thomas Watson relates a similar case. I watched a patient in my ward through an attack of rigor which was distinctly unilateral. Doubtless, in all of these cases, the supply of arterial blood to one side of the brain, although sufficient under ordinary circumstances, became inadequate under disturbing influences—as in syncope—or as in the case of an old native, whom I saw, who had facial paralysis only while he was under the poisonous influence of datura.

Both in India and after they are sent home, English children are very liable to convulsions, especially as an expression of pernicious fever. They are also frequently affected with minor neuroses—twitchings of the facial muscles and hands, nictitation—never, in my experience, pronounced chorea—strabismus, functional and permanent; grinding of the teeth in sleep, night terrors.

Many years ago, attention was drawn in the 'Lancet' to the injurious consequences of children's parties. Children, it was said,

are excited beforehand. They are dressed insufficiently, they amuse themselves into great fatigue, they eat and drink, at late hours, what would try their digestion badly enough in its mid-day vigour; and, worst of all, they lose from two to six hours' sleep. Allowing for differences in climate and custom—in India children's parties assemblable in the day-time—I held these views for many years before I read them. Our children in India generally lead secluded lives, and the excitement of meeting and romping with other children, usually out of the reach of punkahs, almost invariably results in an uneasy night's sleep and fits of sulks or passion, if not worse, upon the following day. Indian parents, are, for the most part, too indulgent to their children, and the need of loving moral discipline for the young is never greater than it is in a tropical climate. Still harshness, not very effective under any circumstances, is worse than useless in the guidance of Indian children.

As it is almost impossible to keep them under the punkah during the greatest heat of the day, these little creatures suffer far more from the climate than their elders do. Etiolated and neurotic to a degree, naughtiness in them is less vice than disease. They must not be angrily chidden, and should never be struck or otherwise severely punished. Indian children are far too much left to native servants, who, at once, over-indulge them, and tease and frighten them. Its mother's side is the proper place for the Indian child.

These principles are equally applicable to men and women in tropical climates. I am confident that, next to vicious excess, loss of sleep is a leading cause of enervation in India. During nine months in the year, the heat in Bengal is so great that our sleep is of bad quality. It may be very well for the subaltern, full of vigour and very moderately worked during the day, to remain until midnight at a dinner-party or until day-break at a ball; but, by the time at which he attains his majority, nature, if not disease and the doctor, will probably suggest to him the necessity of economising his reserve-force either by giving up evening amusements or by taking change to Europe. I recollect hearing an eminent man, who came to India in vigorous middle age, say that he was much disappointed at not being able, when the day's work was over, to do a great deal of his own writing, as John Stuart Mill did. I think that the explanation was this. It is probable that the political economist returned home at five in the afternoon, 'had his tea,' worked in a genial atmosphere until ten, and slept soundly for nine hours. The Indian judge worked hard all day in a hot court; took a long walk when released, and very frequently appeared at late entertainments. This gentleman, being apparently a person of singularly good constitution, is now one of the greatest ornaments of his profession at home; but, unquestionably, he who, even in the very slightest degree, attempts to 'burn his candle

at both ends,' is certain to prove a failure in India, where, quite contrary to the general impression at home, men are, for the most part, extremely over-worked.

I know next to nothing of that 'tropical languor,' except in those who are ill or whose vitality is failing, which our friends in England appear to imagine is almost universal in the East. There, a man with a sound nervous system suffers no failure of aptitude for the very best mental work and for a moderate amount of physical exertion. In him, far from causing low spirits and making him languid, the heat sets up and maintains an exhilarating but very moderate amount of cerebral excitement, tending as little as possible to laziness and blue devils. Still this alacrity is not, of course, to be imprudently presumed upon.

During my long experience as Secretary to the Medical Department,¹ I saw a great many cases of failure of the nervous system under the pressure of '*Office Fag*.' At that time—I believe that now there is a better system—executive engineer officers were especially liable to this affection. Their duties were heavy, demanding very hard work and much exposure out of doors, the charge of large sums of public money, and many difficulties with contractors and workmen. Intemperance was not suspected, as they were all superior men. As regards innervation, these patients were literally 'used up.' They were quite incapable of work, and were not infrequently almost fatuous. In many, memory, even of the commonest facts and dates relating to their malady, was absolutely lost. Usually, these officers, who were mostly young and of good constitution, recovered at home.

Assuredly, the competition system is not a good preparation for an Indian career, in which a sound mind in the sound body of a man of incorruptible honour is the great requisite. Under this system, very few but the very healthiest young men reach India; still, considering that these youths are to be estimated, almost above all others, as 'selected lives,' retrospection of the fruits of the system since its commencement, recalls only too many cases of grave disorders of the nervous system and of early death.

Although the ordinary influences of the climate of India often do not, in many years, much impair the vigour of a sound nervous

¹ In this position, I had an opportunity of gaining a large amount of practical knowledge of Indian disease; as, during six years and a half, I had made myself acquainted with the case of every officer who went home sick or wounded, and read the report of every officer's fatal illness. It was also my duty to study the Annual Reports, &c., of every medical officer at every station in the Presidency of Bengal. Part of the outcome of this course of study was my work on the *Means of Preserving the Health of European Soldiers in India*.

system, one who goes there with an enervated brain will only too speedily discover that the air of the East is in no way bracing. Those with whom the duty of ruling the competitive examination system rests ought to remember that weakened brains are not well qualified for distinguished service in India or anywhere else. They should allow their men to start in life with a fair amount of reserve force.

Doubtless, free tobacco-smoking has a most injurious effect upon the nervous system of many in India. As I have already mentioned, a large proportion of my cases of delirium cum tremore did well; but, latterly, I was always able to prognosticate the death, within twenty-four hours, of a case admitted on a heat-stroke day. The majority of those who kill themselves by hard drinking in India are epileptic. If this be the case in Europe, I failed to observe it in twelve years' large experience of gin drunkards at Guy's Hospital.

It may be said, as a general rule, that, when a disorder of the nervous system is merely functional, early return to Europe, *by long sea voyage*, affords strong hope of recovery.

Cold affusion to the head, by an apparatus similar to that employed by hair-cutters, is most useful and grateful, especially in heat-stroke weather; I had one of these in my European ward. I steadily practised and frequently recommended novel-reading as a means of resting the mind in India.

Return by the overland route, delay in Southern Europe in any but the coolest weather, and change to the Hills are extremely dangerous measures. Mr. J. C. Collins most justly observed, in a report on Darjeeling¹: 'Persons affected with diseases of the brain should not come to Darjeeling for relief, or they will certainly be disappointed; the elevation is too great, and the air too rare: in these cases, headache, and a sense of fullness, and vertigo, are always complained of.' Everyone who has passed up and down the Red Sea, as I have done, and has seen the old Indian passengers sitting with wet cloths over their heads in the saloon apparently wondering who will drop first, will consider that this is a true 'ordeal by fire.' Many deaths at the brain, especially of elderly men, occur every year between Aden and Suez.

I have noticed elsewhere various neuroses prevalent in India. Facial neuralgia, megrim, burning of the feet, asthma, under the head of malarious cachexiæ; false peritonitis, functional gastric difficulties, colic, with diseases of the digestive system; hysteria with the diseases of females; cardiac neuroses are also reviewed.

¹ 'Effects of the Climate of the Darjeeling Hills on the Constitution of Europeans,' *I. A.*, No. 13, p. 1.

Singultus.—Dr. Theobald Ringer reported¹ the case of a native man, with secondary syphilis, who was the subject of very obstinate hiccough. The neurosis gradually abated and ceased on the tenth day. It was treated by chloral, the hypodermic injection of morphia, blistering, and large doses of iodide of potassium.

Mr. T. K. Hall² had under his care, at Rangoon, a native of Madras, who suffered for more than five days from hiccough. He took twelve-grain doses of quinine and recovered.

I believe *Clergyman's Sore Throat*, which not unfrequently occurs in India, to be a true neurosis; and I regard the change in the mucous membrane of the pharynx, which is its usual accompaniment, to be no more the cause of the malady than the attendant bronchitis is the cause of neurotic humid asthma. Still my notes on clergyman's sore throat will be found under the head of *Diseases of the Pharynx*.

Dr. A. Garden has reported³ a series of five cases of *Functional Disease of the Nervous System in native adults*, which will be found valuable by those engaged in the study of Indian neuroses. They are (1) Spinal paresis, partial paralysis, and tremor of the right side. In his commentary upon this case, Dr. Garden remarks: 'There seemed some probability that it was a case of functional nerve disease arising from malaria, which, as a cause predisposing or exciting, stands in so large a proportion to the functional nerve affections met with in this (Saharunpore) district, in which cephalalgia, neuralgia, &c., are more common even than fevers.' It appeared probable, however, that, here, the exciting cause was a 50 mile walk. (2) Headache recurring with vertigo, partial hemiplegia. '(3) Paresis of spinal cord on both sides of motor and sensitive nerves, frontal neuralgia.' '(4) Epilepsy occurring at long intervals during the night, from excessive sexual intercourse, in a man of 50.' '(5) Partial paralysis of the gustatory and hypoglossal nerves, spasm of the sterno-mastoid, sterno-hyoid and sterno-thyroid muscles.'

Indian medical literature is also much indebted to Dr. Garden for an excellent paper⁴ on *Functional Paralysis in Native Children*. This memoir, which is founded upon 31 cases treated in two years in the Saharunpore Dispensary, ought to be studied by every Indian practitioner. The ages of the children were from 15 months to 13 years. In 26 cases, paralysis was the direct and immediate sequel of fever in 18, of small-pox in 7, and of dysentery in 1. Paraplegia occurred in 14 cases, paralysis of the upper extremity in 8, hemiplegia in 4, paralysis of the pharynx in 2, paralysis of leg in 1, tongue 1, left side of face 1. As is usual in native practice, the impatience of friends prevented several of the cases from being watched to a termination;

¹ *I. M. G.* for December 1875.

² *Ibid.* for April 1877, p. 96.

³ *I. A.*, No. 22, p. 384.

⁴ *I. M. G.*, vol. ii, p. 91.

fifteen cases are detailed from Dr. Garden's own notes. Recovery took place in 4 cases of paraplegia, and in 3 the result was unknown. Two cases of hemiplegia recovered, and in 1 the result was unknown. Two cases of paralysis of the upper extremity did well. In one, the result was unknown. Paralysis of one lower extremity, after an attack of fever in a child eleven months old, ended in recovery.

Hugh Macpherson's tables show that, in the eight years ending 1854, the number of European soldiers serving in the Bengal Presidency who were treated for '*Headache and Giddiness*' was 1,219, or three-quarters per cent. of strength, with three deaths or one-quarter per cent. of treated. In the last four years of that term, 67 women, 16 officers, and 2 children were treated for these affections.

Encephalitis and Meningitis.

Macpherson's tables show that, in the eight years ending 1854, the number of European soldiers in the Bengal Presidency treated for inflammation of the brain and its membranes was 76, of whom 15 died, or 10 $\frac{3}{4}$ per cent. During the last four years of that term, 4 officers were treated, all recovered; among the women, there were 2 cases, 1 fatal. In 1882, 8 European soldiers died from these affections. In the native army, there were 9 admissions with 6 deaths. Among the prisoners, there were 18 admissions with 17 deaths.

Save in traumatic cases, I have not seen much of truly inflammatory disease of the brain and its membranes, either at home or in India. There is a good deal of head affection in grave Indian fevers. In the most severe of these, there may be some serous effusion, causing death by coma; but the completeness of the recovery, especially as regards cerebral integrity, in many cases of fever with intense head-symptoms, appears to show that, at least in a very large proportion of cases, this complication is functional—a mere neurosis. I do not recollect to have seen any case in which permanent brain lesion resulted from Indian fever save in one enteric case of my own (such mischief may of course occur in weak brains); this freedom from after ill-effects is by no means characteristic of heat-stroke.

Preparations 782-3-4 in the C. College Museum illustrate various conditions of cerebral meningitis occurring in India.

Delicate as European children are in that climate, I never happened to attend a case of *Tubercular Meningitis* in a child in India. This disease, however, occurs there. Cases of acute hydrocephalus are frequently mentioned in old reports among the barrack children in the Madras Presidency, but it was generally doubtful whether the subjects of this disease contracted it at home or in India. The same question of course holds with regard to the cases noted in

Macpherson's and the Sanitary Commissioner's returns. The former show that, in the four years ending 1853-54, there were, among soldiers' children, in the Bengal Presidency, 53 admissions and 30 deaths, a mortality of 56½ per cent. We find, in the latter, that among the soldiers' children throughout India there were, in 1880, for meningitis, 12 admissions and 9 deaths; none for hydrocephalus. In 1881, the admissions for meningitis were 14, with 7 deaths; for hydrocephalus, 4 admissions and 2 deaths. In 1882, the admissions for meningitis were 19, with 10 deaths; for hydrocephalus 5 admissions with 3 deaths. In 1883, 9 admissions for meningitis with 5 deaths; 5 for hydrocephalus with 2 deaths.

There are two specimens in the Calcutta College Museum. Preparation 785, presented by Tumeez Khan Bahadur, is described as 'brain of a fetus, having fine tubercular deposit like granules of sand in the pia mater, covering the convolutions. This membrane and the shreds of the arachnoid preserved are thickened and opaque.' No. 786, 'Brain of a child, illustrating universal arachnitis. The membrane is thick, dull, and granular. There are small tubercular points in the pia mater.' Morehead states that, during twelve years, in a body of children at the Bycullah Schools, Bombay, numbering from 255 to 350, partly Indo-British, partly of European parentage, mostly ranging in age from 5 to 15, only one case of undoubted strumous meningitis was observed.

Lower Bengal certainly affords resident Europeans great immunity from tuberculosis and struma. In persons moribund from phthisis in my wards, I have frequently noticed, during the last few days of existence, head symptoms—restlessness, extreme irritability of temper, wild staring eyes, almost constant fever, delirium, and fatal coma—which I have traced after death to tubercular meningitis. The Europeans who have thus suffered have brought phthisis to India.

Cerebro-Spinal Fever, see p. 121.

Apoplexy.

Under the head of *Heat-stroke*, I have commented rather freely upon what I regard as the great error of massing together cases of that malady and of true apoplexy in the Indian Medical Returns.

One inconvenient result of this practice is that it is impossible to bring forward any valid statistical evidence to show whether true apoplexy is or is not of frequent occurrence in India. These statistics, however, do not include those classes—the rich, highly-fed, and aged—among whom apoplexy is most prevalent.

I can perceive no grounds of ætiological affinity upon which apoplexy and heat-stroke should be placed together in the *ordo morborum*.

Softening of the brain terminating in apoplexy is common among those rich natives who are over-fed, indolent, intemperate in the use of liquor and drugs, who commit other excesses, and who are often diabetical. I saw a very few cases in East Indians and poor aged natives, and one in a Madras sepoy.

In my long personal experience, I can recall only six unimpeachable and one questionable (embolic?) cases of apoplexy occurring in India in European officers and gentlemen, all of whom were over the age of 50, whose careers in India had been very arduous. In one, death was sudden in an aged man; in the others, life was considerably prolonged. On return home, one of these, a patient of my own, a very self-indulgent man, attacked in middle age, resolutely corrected his mode of living and survived the stroke for eighteen years, completely recovering from right hemiplegia. I examined the brain of a young European of the lower class who died very suddenly of most extensive central cerebral hæmorrhage at a place of public amusement in Calcutta.

I found that *the application of a large mass of ice to the nucha* has great power in arresting stertor. Happening to be in the judges' chambers of the Calcutta High Court, one very hot noon, I was called to see an aged barrister, of free habits, who had fallen in a fit. He was in apoplexy and quite insensible, his face was congested to a plum colour, and there was loud stertor. I had him supported in Marshall Hall's position and applied ice freely to the back of his neck and occiput. The stertor immediately abated and speedily ceased. He recovered completely, without paralysis, in a few days, but died in court about two years subsequently. A native with carotid aneurysm fell stertorous, an embolus having become detached from the sac. Ice to the nucha immediately checked the stertor, but he died. My nurse had a supply of ice constantly ready to be used in this manner, especially in cases of epilepsy, which are generally much relieved by the application of ice. I saw stertor, with perfect consciousness, in an apoplectic native, and heard of another similar case.

I had repeated experience of the utility of placing, raised in Marshall Hall's position, insensible apoplectics whom I found suffocating on their backs with their heads fallen back.

Abscess and Gangrene of the Brain.

Allan Webb gives (p. *225) three very important cases of acute abscess or gangrene of the brain in natives: (1) From the effects of injuries received in a parturient woman from native midwives; (2) From hernia; and (3) From gangrenous dysentery. Here Webb clearly recognised the occurrence of ichoræmia.

A very interesting but imperfectly reported case of abscess of the pons will be found in No. viii., p. 637, of the 'Indian Annals of Medical

Science. An officer, aged 22, of strumous habit, addicted to excessive smoking, and a free drinker, arrived from the district January 31 with incomplete loss of voluntary motion on the left side; sensibility not much impaired. Drags the leg after him in walking; mouth drawn to the right side; tongue, when protruded, inclines to the same side. Impaired memory and diminution of the intellectual functions. Speech, indistinct. Answers in an abrupt and angry manner. Head hot. Countenance firm and flushed. Pupils dilated. Urine passed with difficulty. Bowels confined. Pulse hard, about 100. Quite conscious; fancies he has only a rheumatic attack. Feb. 1st, dizziness: urine had to be drawn off by catheter; speech very indistinct. 2nd, Evidence of paralysis increased; speech muttering and indistinct; vision, disturbed; *vesp.* tongue paralysed. Is quite conscious and only complains of slight pain in the *back* of the head. 3rd, 2 P.M., cannot swallow: is sleeping and breathing stertorously and puffing the cheeks. 4 P.M. *breathing stertorously but conscious*, cannot swallow the pills. 4th, Dysphagia complete; says his nose and throat are stuffed with mucus. 11 A.M. Breathing hurried with mucous rattle in the throat, conscious, complete dysphagia. At noon, said that he was dying. Became comatose and died at half-past one. The brain and cerebellum were healthy. On dividing the medulla oblongata, there was an escape of about two drachms of pus. The lesion existed in the pons Varolii, which 'was in a decided state of non-vascular ramollissement, and at the base' (?) 'a cavity not encysted, which contained, in all, about three drachms of thick pus of recent formation.' The context renders it almost certain that the lesion was an abscess originating in the pons, although the reporter's obscure language leaves the fact questionable.

In the Calcutta College Museum, Prep. 794 displays what appears to be a chronic brain abscess, about as large as a small pigeon's egg, 'surrounded by a dense wall of fibrous structure; 795 shows the cavity of a small abscess in the middle lobe of the left hemisphere,' and 796 exhibits 'abscesses in both hemispheres.'

I saw in India several cases of *Abscess of the Brain* from heavy blows, as by a brick or a bamboo *lattee*. Here, except in a case where extensive fracture of the skull opened the middle meningeal artery, and in which I trephined (powdered alum arrested the very active hæmorrhage), the symptoms commenced with violent rigor at about the fifteenth day after receipt of the injury, necrosis of the skull having set in. In most cases of this kind, we notice a more or less distinct line of demarcation between the healthy skull and its dead portion. In the case of a very young native woman at Chittagong, the separation, tending to removal, of a portion of the frontal bone was so distinct as to suggest the possibility of spontaneous trephining.

My own experience alone would enable me to say a good deal re-

garding *Injuries to the Head*, were not my present business confined to conditions of disease peculiar to India. But I may just allude to the following. A poor Cookie, from the hill districts east of Chittagong, brought his murdered child in a sack, carried over his shoulders, to the magistrate. The body was so much decomposed, and the calvarium so extensively smashed, that the large bones fell out when the scalp was divided. It was evident that a very heavy blow had fractured the bones, but they were exceedingly friable, being almost universally involved in an enormous vascular nævus which, in parts, rendered them nearly an inch thick. In a subject in our dissecting room, an iron brad, about an inch long, appeared to have remained for years piercing the frontal sinus and falx, standing like a pier of a bridge in the current of the sinus apparently without causing the slightest disturbance.

In the calvarium, not in the site of a fontanel, in a very aged native woman, who died of ordinary disease in my ward, was found an oval deficiency of skull, as large as the bowl of a tea-spoon. The edges of bone, without diploe, were nearly as sharp as a knife all round the aperture, which was closed by a thick membrane, which resembled, but was not, *dura mater*, from which it was separable. I trephined a cooly (native labourer) whose frontal bone was very extensively smashed by the hook of a crane which struck it, in the mesial line. This and some other injured skulls which I have examined were so unsymmetrical that, if I had opportunity, I would search in large collections for other ill-formed skulls which had received fatal injury, and I would also search the accident wards of hospitals for persons with deformed skulls. The idea may appear fanciful, but it is certain that the instinct of self-preservation is far more highly developed in some persons than in others, and I believe that avoidable 'accidents' occur most frequently to those in whom it is least vigorous. Watch the action of people who are surprised by the rush of a run-away horse: one stands panic-stricken right in the animal's course and is knocked down, another tries to cross its track and falls under its hoofs, a third attempts to run before it as if pursued, a fourth leaps skilfully aside, follows the animal and catches the bridle.

An intelligent little East Indian boy, at Chittagong, fell out of a verandah about eighteen feet high, upon a heap of bricks. His vertex was the only part severely injured. I had never seen so enormous a 'thrombus' as that which existed over his crown. Although familiar with minor injuries of the kind, I thought, as my fingers sank in, that a hand's breadth of the skull was smashed; but, as he appeared rather pleased with his condition than otherwise, I merely applied a lotion, and 'looked out for storms.' He recovered in a few days without any evidence of injury to the skull or a single bad symptom. I, many years ago, saw a far more remarkable illustration of the trite observa-

tion that the young are often tolerant of severe injuries to the head, which is balanced by the fact that what appear to be very trivial injuries to the heads of children, especially the delicate and strumous, often set up fatal disease. A little girl lived for several days with a fracture which, had it been perfectly circumferential, would have taken off the calvarium. As it dipped far down at one end, its length was considerably greater than that of a string carried evenly round the skull above the ears. A very fine and powerful sea-faring man cut an opening two inches behind his chin, and discharged a pocket pistol loaded with a bullet through his tongue up into his head. He remained alive nearly three days, lying in what appeared to be a state of coma. The comrade who watched him during the second night declared that P. got up and stood looking about, and when asked 'What do you want?' replied in a tone of anger, 'What do I want!' He then lay down and again became insensible. The ball was found just below the sagittal suture. I long kept it; a strong fragment of bone, which it carried with it, had cut it half through. I met with several cases in which it appeared unquestionable that recovery followed fracture of the base of the skull.

On the Calcutta drive, I saw a bulky merchant in the prime of life thrown from his buggy. He fell in a sitting position. I took him to his hotel, and remained with him until his own medical man relieved me. He had evidently received a very severe *Shock* to the cerebro-spinal system, but appeared perfectly conscious throughout; he, however, quite forgot that I had driven him home.¹

I observed and made notes of cases of *Shock with Excitement* many years before I saw this condition described in text-books. A middle-aged clerk, of wiry frame, was brought to Guy's Hospital immediately after his pelvis had been crushed by a waggon wheel in the Borough High Street. He had been drinking, but, we were convinced, was not merely intoxicated. There were all the signs of ordinary collapse, save that, although he was dying rapidly, he was extremely violent. At Chittagong, a tigress, which lay concealed in a garden, was attacked by a party of Europeans and natives. The animal making a rush, one of the latter, a professional *shikarree* (hunter), had the outer half of his knee-joint removed at a snap. He was a very courageous and strong little man of mature age. He was wildly violent and would listen to none of our arguments, that he would probably recover if he would allow me to amputate, although a poor fellow, convalescing from amputation of the thigh for crocodile-bite, was led to his side. Every attempt to approach him enraged him beyond measure. He continued, almost until he died, to dash the mangled limb down upon the *tukta* (wooden bed frame), scattering the blood in all directions, a ghastly sight even to a surgeon.

¹ This forgetfulness was undoubtedly genuine.

HEAT-STROKE.

Persons whose nervous systems have been shaken by disease, excess, or traumatic injury are extremely liable to be attacked with heat-stroke.

The following case is one of scores that might be cited. A young officer suffered in India from a very severe injury to the skull, for which he took leave to Europe, and appeared to recover. On his way back to India, he landed at Aden, and, most imprudently, walked to the Cantonment. He died with heat-stroke symptoms. In England, even in cold weather, some old Indians are unpleasantly affected by strong sunshine.¹

One who has suffered from any affection of the nervous system in the East, ought to be most careful to seek a cool climate, and not to expose himself in hot weather at home. In March 1882, the Paris correspondent to the 'Daily Telegraph' wrote as follows: 'The first case of sun-stroke recorded this year occurred on Sunday, the victim being, curious to relate, M. Jules Blancsube, the Deputy for Cochin China, who had only been in France a few days. M. Blancsube is extremely unwell, but hopes are entertained that he may recover. *A case of sun-stroke in Paris in the month of March, the sufferer, moreover, being accustomed to a tropical climate, is, I need hardly add, a very rare event.*'

The italics are mine. It can scarcely be doubted that, here, the attack was due to impaired innervation consequent upon residence in a tropical climate.

About a year ago, I heard of two healthy middle-aged ladies, long resident in India, who suffered from heat-stroke after returning to England.

It would be well if all persons—the strong as well as the enfeebled,—in temperate as well as in tropical climates, in cool as well as in sultry weather, could bear in mind the fact that unusual exertion in the direct rays of the sun is not without danger. Thus I read,² in an account of the march of our volunteer battalions upon Lydden, near Dover, on April 24, 1886, that 'a good many of the infantry found the heat, the dust, and the sustained exertion too much for them. Despite a cool easterly wind, the temperature in the sun was high, the dust was excessive, and marching in the closed-up ranks became trying in no slight degree. As a consequence, falling out grew unpleasantly frequent, while, to make matters worse, no ambulance

¹ Suetonius wrote of Augustus Cæsar: 'His body being weakened and shattered, he could scarce endure extremity either of heat or cold. He could not endure so much as the winter sun.'

² *Daily Telegraph* for April 26, 1886.

waggon were in close attendance, the whole of these vehicles being in the rear of all; an arrangement which, as regards one or two cases of epilepsy, was productive of great inconvenience and anxiety.'

The subject of heat-stroke is one of such great importance, and its Indian literature is so valuable, that I would strongly recommend the perusal of the below-mentioned memoirs to the young physician.¹

In adhering to the two designations 'Apoplexy' and 'Sun-stroke,' and by entering cases under either head, the Indian Medical Returns have long rendered the statistics of heat-stroke as obscure as possible.

Macpherson's Table shows that, in the eight years ending 1853-54, the admissions for *Apoplexy*, among European soldiers serving in Bengal, were 829 or $\frac{1}{2}$ per cent. of strength, or 12 times more numerous than in England, but the proportion of deaths so treated was larger at home than in India, viz., $77\frac{1}{3}$ to $67\frac{4}{5}$. Of *Sun-stroke*, there were 38 cases and 7 deaths, or $18\frac{2}{5}$ per cent. Mr. Macpherson added, 'We believe that many of the cases returned as "*Apoplexy*" were, in fact, cases of *Coup de Soleil*, or, as the French more correctly term the disease, *Heat-stroke*.' It appears very singular that, during the 26 years which have elapsed since this remark was made, this confusion of terms should have been allowed, and that no medical authority should have succeeded either in placing together conditions which are identically the same, or in widely separating two states which have no ætiological feature in common.

In his report for 1880, p. 31, the Sanitary Commissioner gives a table which 'shows the prevalence of apoplexy and sun-stroke by stations

¹ VAN SOMEREN. 'Pathology of *Coup de Soleil*,' *Hyderabad Medical and Physical Transactions*. It would be useless to search for this valuable publication, but a full abstract of the paper will be found in *I. A.*, No. 2, p. 738.

MOREHEAD. Chap. xxvi., on 'Sun-stroke.' At p. 605 Morehead gives twenty-four references to important papers on this affection.

MARCUS HILL. 'Remarks on the Disease termed Insolation or Heat Apoplexy, with Observations on its Pathology,' *I. A.*, vol. iii. p. 188.

J. B. SCRIVEN. 'On Sun Fever,' *Ibid.* vol. iv. p. 496.

J. R. MARTIN. 'On Heat Apoplexy, &c.,' *Lancet*, vol. ii. 1859.

T. LONGMORE. 'Heat Apoplexy,' *I. A.*, No. 12, p. 396, and *Lancet*, vol. i. for 1859.

DR. JAMES P. BROUGHAM. 'Heat Apoplexy,' *I. A.*, No. 12, p. 526.

W. J. MOORE. 'On the Different Varieties of Insolation,' *Ibid.* No. 24, p. 241.

W. CURRAN. *Sun-stroke as Seen in India*, 1880.

J. FAYRER. 'Sun-stroke,' *Practitioner*, 1876. 'Insolation or Sun-stroke,' *Transactions of the International Medical Congress of 1881*, vol. ii. p. 554.

J. FAYRER. Art. 'Sun-stroke' in Quain's *Dictionary of Medicine*.

J. FAYRER and J. EWART. *Du Traitement des Maladies Tropicales dans les Climats Tempérés*, p. 20. *Des Conséquences de l'Insolation et de la Fièvre Thermique*, 1883.

For remarks on the Use of Quinine in Heat Stroke, see *I. M. G.*, vols. iv. No. 7 (Waller) and v. Nos. 3, 6, and 12; vol. vi. Nos. 4, 6, and 7; *Ibid.* for 1877, p. 193 (Francis).

and groups.' In his report for 1882, p. 31, he amalgamates these two conditions under the common designation of 'Apoplexy.' As I am unable to recognise the utility of tables thus constructed, I shall merely cite the figures from the returns for 1882 and 1883, in most of which the two diseases are inextricably cast together. Thus, within the first year, 48 *European soldiers* are shown to have died in India from 'Apoplexy and Sun-stroke.' With these is bracketed a death from 'Cerebral Hæmorrhage,' as if sun-stroke and cerebral hæmorrhage were not as distinct and as absolutely separable entities in disease as tetanus and hydrophobia or diarrhœa and dysentery are! For 'Sun-stroke,' 8 *Euro-peans* were invalided. *Women*—6 were admitted under 'Apoplexy and Sun-stroke,' of whom 2 died. *Children*—3 admissions, 1 death. In the returns for *Native Troops*, there are separate headings, 'Apoplexy' and 'Sun-stroke,' the admissions being kept apart and the deaths being bracketed together. There appear to have been 18 cases of 'Apoplexy' and 22 of 'Sun-stroke,' out of which 40 cases 17 proved fatal. In like manner, 19 *Prisoners* were admitted for 'Apoplexy,' and 17 for 'Sun-stroke.' Among these 36 cases, there were 18 unclassified deaths.

So also, in the return for 1883, apoplexy, sun-stroke, paralysis, hemiplegia, are bracketed together, the admissions being shown separately, and the deaths being massed inextricably. Considering that these returns are generally clear and well-constructed, it appears hard to understand why deaths from sun-stroke should not be set apart from those in which hemiplegia has been a symptom. Seeing that every medical man in India is highly capable of differentiating his cases of true apoplexy from those of heat-stroke; and that, at least during the last quarter of a century, every medical officer has been perfectly well aware of the fact that nearly all the cases returned under the heading 'Apoplexy and Sun-stroke' are, in reality, cases of the latter disease, it is, I consider, much to be regretted that, while one man correctly returns his case as 'Sun-stroke,' another should have it at his discretion to designate his case of the same disease, very unscientifically, as 'Heat-Apoplexy,' or, less accurately still, as 'Apoplexy.'

I agree with Mr. Macpherson in thinking that the designation 'Heat-stroke' is as free as possible from objection, and that its general adoption would be a step in advance. Seeing that patients in hospital have been frequently attacked, and that cases have occurred in the night, the terms 'Insolation' and 'Sun-stroke' are by no means universally applicable.

The observations of Dr. Furnell and others have shown that the temperature of healthy Europeans maintains its normal standard in tropical climates; and it is certain that men of good constitution, who perspire freely, can endure an amount of heat, as in the stoke-rooms of Indian steamers, very far higher than that which prevails in heat-

stroke weather. There is, doubtless, a point of heat upon exposure to which all human life would instantly perish, but no climate is subject to such heat. The aboriginal inhabitants of tropical climates are, as long as they remain in unbroken health, capable of working in the greatest heat, under which, due care being taken, Europeans are not prostrated. In the discussion which ensued upon the reading of Sir Joseph Fayrer's paper on 'Sun-stroke' at the International Medical Congress of 1881,¹ referring to the fact that the majority of the native inhabitants of India, both Hindus and Mohammedans, being the descendants of Caucasian foreigners, are liable to this affection, I inquired whether the aboriginal Mongolian people of Hindustan are subject to it. I had seen a good deal of them, and believed that they are not. This elicited from Dr. Yandell, Louisville, Kentucky, the observation that the negro suffers much less than the white from sun-stroke. The negro bears the sun's heat better than the white man. Whether this is due to what is supposed to be the greater activity of the negro's skin, he was not prepared to say. The negro who works in the harvest field and in the cotton field very rarely suffers from sun-stroke. When he removes to the city, and there lives in crowded houses and close rooms, is subjected to the same influences which demoralise the white man, and becomes loose and irregular in his habits, committing excesses in eating, but more especially in drinking, he then becomes liable to sun-stroke. Yet the negro remains, under even these circumstances, better able to withstand the evil effects of heat than the white man working by his side. Throughout my long residence in India, the question 'Can one harden oneself against tropical heat?' was ever present in my mind. The impression which my experience has left is distinctly in the negative. True, a large proportion of those who are attacked with heat-stroke are young and apparently healthy men recently arrived in India; but, for the most part, these are victims of excess and recklessly imprudent exposure. Long residence in India does not acclimatise us, but it teaches us how to live in the country, and how to avoid most of the dangers of its climate. Long residence in the tropics does not strengthen our brains against heat-stroke, but no one is more secure against its attacks than an experienced prudent Englishman, still young and with a well-preserved brain.

I have given a brief account of the principal causes of heat-stroke in my chapter on the *Effects of Indian Heat in the Production of Disease*, page 11, and in the remarks on *Ardent Fever*, page 215.

Study the constitutional causes of heat-stroke, is a maxim which I offer to the young Indian physician. 'Why,' it may be said, 'heat-stroke is a mere accident to which all are equally liable! You might as well suggest an inquiry into the constitutional causes of dog-bite!' I reply, No—supposing that Sol struck down nine-tenths of the men

¹ *Transactions*, vol. ii. p. 500.

of a detachment (I do not know of any instance in which so large a proportion has suffered), we should find, on close investigation, that he had been remarkably discriminative in the selection of the most unfit.

Undoubtedly, he will learn most with regard to the ætiology and prophylaxis of heat-stroke who looks most searchingly into *the previous constitutional state* of each individual sufferer. Allowing that no man however healthy, can endure, without falling exhausted, unlimited fatigue in heat-stroke weather, I believe that ninety-nine per cent. of those who thus fall are previously diseased or weakened. Hence careful analysis of upwards of two dozen cases of so-called sun-stroke would probably discover that :—

1 is the subject of alcoholic poisoning.

2 has over-eaten himself and has then gone out in the sun-glare.

3 has not slept for a night or two.

4 has epilepsy excited by heat, not heat-stroke.

5 has cholera.

6 has diseased liver or kidneys, and is the subject of cholæmia or uræmia, or of both combined.

7 has brain disease.

8 has a fatty or otherwise weak heart.

9 has malarial fever, the hot stage occurring on a sun-stroke day [see 'Ardent or Heat-stroke Complication of Paludal Fever,' pages 168 and 188]. Shakespeare understood this—

An ague subtly taints

Even then when we sit idly in the sun.

Troilus and Cressida.

10 is old enough to have faulty cerebral arteries, as was the case with William Cullen Bryant, who, at the age of 84, was struck down by the sun while speaking bare-headed in an open place.

11 has fainted, perhaps to death.

12 is enervated by tobacco-smoking.

13 by raking.

14 by excessive tea or soda-water drinking.

15 has a slight scorbutic taint.

16 has syphilis lurking in his system.

17 has previously suffered from sun-stroke or meningitis in fever.

18 sustained long ago a severe injury to the head.

19 has diarrhœa.

20 eats opium.

21 has had exhausting duty of late.

22 is the subject of 'office-fag.'

23 has recently sustained deep mental anxiety or great affliction.

24, an English country gentleman, with whose case I am ac-

quainted, at the age of 48, took active part in an election—work which was new to him—he died with symptoms of acute cerebral inflammation shortly after having spoken on the hustings with his hat off in the sun-glare—he was an amiable man, but lunacy prevailed in his family; and, soon after he arrived at maturity, he was under confinement as a maniac for eight or ten years.

25 is a man whose constitution bears close investigation, the vigour of whose nervous system appears to have succumbed to excessive heat. This 25th case, however, is evidently less explicable than the rest, and I think it most probable that, if we could sift it thoroughly, we should find that the man had been previously subject to some valid cause of enervation.

Numerous as the constitutional causes of heat-stroke are, all Indian experience combines to show that Drunkenness is the chief. This used to be notoriously the disease of canteen sergeants, who had little work and unchecked access to ardent spirits.

The following is the latest illustration of hundreds that I have seen.¹ ‘ Surgeon-Major Beattie, in medical charge, arrived at Suez from Aden on August 8. He reported that, “before leaving the docks, the men got some bad brandy, which, aided by a powerful sun, knocked down ten of them, on the short march of three miles to the Victoria Hospital. One actually died on the way; another in hospital. Two days after this, a third death from stroke of the sun took place; one died of diarrhœa.”’ Dr. Smith says, ‘All the fourteen cases of sun-stroke were in the Seaforth Highlanders,’ . . . ‘the rest of the cases’ [four] ‘occurred on August 20. There was no other case of sun-stroke in the force, although it was exposed to a burning sun during the whole of September 13, and most of the 14th, before the tents arrived at Zagazig.’

I have never seen or heard of extravasation of blood, or true apoplexy, in heat-stroke; but, doubtless, he who has a tendency to true apoplexy places himself in imminent danger when he ventures out on a heat-stroke day. On the occasion above alluded to, Sir Joseph Fayrer and those who discussed his paper said much that is practically important regarding the *After Consequences of Heat-stroke*. He observed that, though many who are attacked recover, or rather do not die, a large proportion are permanently injured and become invalids for life, which is frequently shortened by obscure cerebral or meningeal changes that affect the sufferer in various degrees of intensity: irritability, impaired memory, headache, mania or dementia, partial or complete paraplegia, partial or complete blindness, intolerance of the sun’s rays, rendering a person, otherwise fairly healthy, incapable of serving in hot climates or often daring exposure to the

¹ Deputy Surgeon-General Colvin Smith’s *Report of the Indian Division, Egypt Expeditionary Force, 1882, S. C. R., 1883, p. 193.*

sun; it may be, gradually ending in fatuity, dementia, or epilepsy, perhaps combined, chronic meningitis, with thickening of calvaria and intense cephalalgia, or, in a lesser degree, in disordered innervation and general functional derangement which seriously compromise health. Dr. Christie, after some years' experience of sun-stroke, was satisfied that its after-effects are not sufficiently understood. In a large number of cases, after an attack, the mental capacity is so far interfered with as to render a man quite unfit to ever return to a hot climate. The pathological changes fully bear out Sir Joseph Fayrer's views. In three or four cases, he has met with remarkable growth of spiculæ of bone from the skull, with thickening and opacity of the membranes. In one case, epileptiform convulsions with complications of mania followed. Dr. Yandell said that he did not remember to have seen, in America, a genuine attack of sun-stroke from which the individual ever perfectly recovered. Some twist, some evil remained. The patient's physical strength, his mental force, his moral powers were lessened or altered.

Treatment.—I think that I have made it evident that, to treat a case of heat-stroke fairly, we ought to be acquainted with the predisposing cause of the attack. In a regiment, the medical officer may frequently possess the clue, being well acquainted with the men's health histories.

When there are friends about the sufferer, a few brief questions—Does he drink? Has he lately been ill? Does he eat opium? Is he liable to fits? Has he been over-worked of late?—will often aid us; while jaundice, great debility, collapse, a faulty heart, an enlarged spleen, diseased liver or kidneys, old age, scalp cicatrices, the odour of alcohol or tobacco, the presence of a syphilitic or scorbutic taint, will speak for themselves.

The first thing to be looked for is the characteristic dry, pungent burning heat of the surface of the body and the exceeding high temperature. The patient with a pallid countenance, a feeble pulse, and a cool or cold surface moist with perspiration, has either fainted or is in collapse—is the subject of probably deadly shock. In either case, to place him in an ice bath, or to allow a heavy douche to fall upon his head, will be, especially if the heart be faulty, to 'put him out.' Even when the temperature is at the very highest, we must recollect, in applying the cold which is here so needful, *to be guided by the strength of the pulse.* The spark of life is here so feeble as to be liable to extinction under the depressant action of extreme cold long continued. All our military surgeons relied greatly upon the efficacy of a stream of water from the bheestie's *mussuck* (sheep's skin). I, latterly, always employed 'planing' the head with a slab of ice. In the discussion cited above, Dr. Joseph Ewart, of Brighton, called attention to the importance of employing cold iced baths in the treatment of

heat-stroke, for the purpose of reducing the temperature of the body. At the General Hospital in Calcutta, he had seen patients thus restored from profound insensibility to consciousness in a few minutes. He insisted that this bath is only applicable where there is augmented temperature.

Chronic Hydrocephalus.

I have seen, in European children in India, a few moderate cases, not more marked than that of De Quincey, the English opium-eater, whose portrait shows that he was distinctly hydrocephalic, a fact which throws light upon some of his mental peculiarities.¹

This disease is rather frequently seen in natives, because those afflicted in this manner are well qualified either to beg or be begged for. Some very remarkable hydrocephalics may be seen, from time to time, in the large body of mendicants who infest the streets of Calcutta. I used to show, in my lectures, an immature hydrocephalic cranium which, in maturity, would have rivalled that of Cardinal in the museum at Guy's. Morehead saw only two cases of chronic hydrocephalus in India.

In a Hindoo male infant, 6 months old, Dr. L. A. Kidd found that the circumference of the head was 20 inches; the transverse measurement was 16 inches; the fontanels very distinct. There were strabismus and continual rolling of the eyes, loss of appetite and great restlessness. Treatment by medicines and strapping having failed, puncture was made at the anterior fontanel with a small trocar. The removal of twelve ounces of fluid appeared to cause faintness. Strapping was re-applied. The child was considered to be likely to recover 23 days later, when the report was made.² I saw *Meningocele* in a native infant.

I have more than once met with cases of *Gummata and other Tumours of the Brain*. (See the chapter on Aphasia, p. 410.) Dr. Duncan Stewart brought before the Medical and Physical Society of Calcutta³ the case of a military man, *æt.* 47, subject to low spirits, frontal headache, and intolerance of strong light. He was fat and lethargic. There was no paralysis or convulsion. He gradually sank into a nearly comatose state which lasted for about three weeks, the animal func-

¹ A series of observations upon the mental condition of the subjects of moderate hydrocephalus would form an interesting chapter. I must have been a boy of thirteen or fourteen, when a charming little fellow of nine, handsome but decidedly hydrocephalic, entered my class at school and 'took down' every one of us. Although he was very bright, promising to become an admirable scholar, his pronunciation was still as childish as it must have been at the age of three. Dr. Georges Herve's inquiry into the *post-mortem* condition of Cuvier's cranium and brain, clearly shows that the great naturalist was the subject of hydrocephalus in his childhood.—*Revue de Thérapie*, December 1, and *M. T.*, December 29, 1883.

² *I. M. G.*, vol. i. p. 184.

³ *Transactions*, vol. viii. part 2, p. 142.

tions continuing unimpaired, voluntary motion perfect, circulation undisturbed, temperature natural. Pupils sensible to the light to the last. An *osteo-fibrous tumour* was found between the lobes of the cerebrum, near the origin of the frontal sinus, weighing 35 grains, about the size of a large pigeon's egg, divided longitudinally, its flat surface adhering firmly to the falx cerebri, close to the crista galli, but not connected with it; its free surface covered by pia mater; the apex indented by the pressure of the neighbouring sinus. The fatal symptoms were accounted for by meningeal congestion, and great sub-arachnoid effusion of serum.

Dr. Drake-Brockman has published¹ a valuable commentary on two cases of *malignant and scrofulous disease of the sphenoid bone*. The mischief was developed in the cancellated structure of the bone.

Malformations.

Prep. 990 in Ewart's Catalogue is an anencephalous foetus, probably native. Prep. 988 is 'A foetus in which the convolutions of the brain are undeveloped, and in which there is spina bifida. There are scarcely any frontal or parietal bones, the flat surface of the cranium being situated on a plane about half-an-inch above the eyebrows and the ears.'

There was a European child in Calcutta which, judging from a portrait taken at about the age of two years, was pretty and fairly grown; but the cranium was frog-like, and so deficient in its arch as to give the idea of a degree of *Anencephalus* as extreme as is consistent with extra-uterine existence. An officer's family, of apparently good ability, had such remarkable deficiency of anterior cerebral lobes, with projecting eyes, as to constitute a singular deformity in persons of so much intelligence. In the father, this deformity was marked, but considerably less.

What would have been a very interesting case—had I been allowed opportunity of examining it fully—of congenital unilateral deficiency of brain (*Atelencephalia*) was in my ward for a day or two suffering from some ordinary illness—a respectable lad of 17, slightly dark. The right side of his head was so very markedly deficient that none of the upper portion of that cerebral hemisphere could have been fully developed, and there was probably considerable arrest of growth of cerebellum on that side. On the left side, the head was small, but well formed. Generally, he was ill-grown and of delicate appearance. The left limbs were paralysed and contracted, and would have been considered atrophic had they belonged to a child of three. The countenance was natural and pleasing. He appeared quite intelligent, and

¹ See *I. M. G.* for November 1885.

saw and heard well. I fear that the unusual notice which he received caused him to leave precipitately.

There are very full illustrations, noted (pages 26, 51, and 118) in Dr. Sibthorpe's catalogue, of the condition of a woman (doubtless native) whose body was brought to the Madras Medical College dissecting room. The *right* cerebral hemisphere, *crus cerebri*, *corpus albicans*, and optic nerve, were atrophied. The *left* cerebral and the *right* cerebellar hemispheres appeared normal. The *left* lobe of the cerebellum was atrophied. The cranial bones were thicker than usual, and there was hypertrophy of the diploë which corresponded with the atrophied brain. The right half of the tongue was one-half smaller than the left. The upper jaw on the left side held nine, and the lower on the same side, eleven teeth. On the other side, the jaws (as far as the mesial line) were devoid of teeth [age not given]. The left chest was only half as capacious as the right, and the left lung was considerably smaller than the right. The left ribs were less developed than the right. The left half of the pelvis and the upper and lower limbs of that side were less bulky than those on the other side.

Disease of the Cerebral Arteries.

A high-caste Brahmin became a minister of the Church of England. He, when considerably advanced in life, suffered great hardships during the Mutiny. He was afterwards attacked with a most distressing, constantly present, sound in the head, on account of which he came down to Calcutta, and consulted most of the medical staff there. On applying the stethoscope to any part of the cranial vault, there was heard what was evidently a systolic bruit. I can compare it to nothing but the sound of the most delicately-toned bell, sharply but slightly struck by a metallic point. One could imagine that the cranial vault was a silver cup, and that each beat ran around it in a prolonged thrill of the finest resonance. I can produce a similar, but far coarser and less prolonged vibration, by striking the edge of a hand-bell with an ivory pen-holder. Pressure upon the left common carotid stopped this at once; but, although the annoyance was very great, his general health was so good that it was not thought advisable, at his age, to tie the artery. He lived for some years subsequently. There was no *p.-m.*

Under the head of *Cerebral Neuroses*, I have alluded to the prevalence of minor functional and transient conditions of *Aphasia*, *Amnesia*, and *Agraphia* in those who overwork their brains in a tropical climate. It has been said that great men sometimes affect to 'call Jack Tom, and Tom Harry.' This is certainly not infrequently the case, but it must be recollected that great men are very often old men. This was one of the early manifestations of failing cerebration

in Linnæus. There were, in India, two very eminent personages in whom this practice went almost to the length of eccentricity. One of these did useful work, and lived for several years after his retirement to this country. The other has long survived his return, and has married. Their native air evidently in great measure restored the natural hardness of their heads.

I met with several marked cases of *Aphasia* in India. A distinguished Calcutta surgeon lived into advanced old age with right hemiplegia. Of late years, he would read much, but did not speak to visitors. On one occasion, a clergyman called upon him, and, only receiving a look of recognition, said, 'You do not recollect me, Dr. —.' With another intelligent look, the old man turned over the leaves of his book and placed his finger upon a passage in which his questioner was mentioned.

I had opportunity of watching two of the three typical cases so graphically described by Sir Joseph Fayrer¹: That of S—, a well-born European, lamentably 'his own enemy,' whom I had repeatedly treated for other diseases, in whom an apparently slight injury to the left side of the head ended in circumscribed necrosis of skull and an abscess of the brain about the size of a hen's egg, extending forwards over the island of Reil, with right paralysis; and that of Mr. —, whom I had long known, and in whom there was probably temporary embolism of the left middle cerebral artery. He recovered, except that he occasionally forgot a word. He was seen walking in Regent Street, but died in about 26 months after the attack. In S., when the symptoms of cerebral pressure were gravest, before the trephine was used, and latterly when they were most grave, there was complete aphasia; when they were lighter, he could only utter sounds like 'ai cairna,' which he repeated incessantly. Improving, he could say 'pipe' when he wanted one, then 'I had padsum' for spasm. When his state temporarily improved, his speech was 'very fair, though slow, as if it were an effort of memory to find the words, or that the tongue required schooling to articulate them. Occasionally he hesitated at a word, and, failing to pronounce it, substituted another, not always relevant.' Towards the last, before he became speechless, he howled when he wanted anything. To the last, he could be made to understand, although intelligence was much affected. Before the trephine was used, there being complete aphasia, he endeavoured to express what he wanted in writing with his left hand; he could only trace his name, his mind seeming clear. The servants of Mr. — said that he was lying on his couch in the morning, when they suddenly saw that he was convulsed on the *right* side, and that he was quite unconscious. There was no hemiplegia subsequently. It is noticed that, on the third day, his appearance being quite intelligent, he could reply

¹ *Observations*, pp. 609 and 626.

to a first simple question, such as 'Have you slept or eaten well?' answering 'Oh yes,' or 'No,' as the case might be; but the next question, however simple, puzzled him completely, and the reply, for he tried to answer, was the most incoherent nonsense—words without connection or meaning. Having long been well acquainted with the expressions of his animated and intellectual face, I was much struck by the fact that, while he evidently understood, with courteous attention, everything that was said to him, his inability to give an answer at any length did not appear to cause him the slightest embarrassment or vexation. On the third day, when his powers of expression were at the lowest, he could only, on being asked to write a note, scrawl the figure of 8, saying: 'I can't write; oh, that's just it.'

It is noteworthy, as bearing upon Dr. Francis' observation cited below, that aphasia may arise from paludal influence, that this gentleman served long at the very malarial station, Chittagong, while I was medical officer there; and that, there, one of his daughters died of malarial eclampsia.

In 1836, Mr. E. F. Downes published¹ a case which closely resembled that of Mr. ——. An English (?) gentleman, at Neemuch, perceived a numbness and slight tingling sensation in the *right* hand, with loss of muscular power, his articulation became extremely indistinct, and, before he could finish a sentence, he appeared to have forgotten what he wished to say. He denied that he had headache; the pupils were exceedingly contracted and sluggish. The rapid abstraction of 20 ozs. of blood appears to have been followed by cessation of these symptoms; but, something occurring to ruffle his temper, he was again seized with loss of memory and difficulty in utterance. He made great efforts to speak. He replied with much hesitation in monosyllables, quite at variance with the questions put to him. He was restless, walking from one room to another, as if he had some object in view. He appeared to understand questions, but to be incapable of answering. When asked to describe his feelings on paper, his hand was so tremulous that he could only dip the pen after several efforts. He succeeded in writing 'My dear,' and then commenced with a scrawl. Making several attempts to proceed, he threw the pen down, shook his head, and ejaculated: 'No! no!' Bleeding from the temporal artery to 16 ounces was followed by great relief; the pupils, which had been contracted, dilated freely. 'In the evening, he was perfectly free from the complaint.' As the report was made fourteen months after the occurrence, it is to be presumed that, at least so far, the patient did well. It is particularly noticed that, previous to the attack, the patient had suffered 'first from remittent and afterwards from intermittent fever.'

In 1867 Dr. Francis reported² a case of aphasia, which appeared

¹ *I. M. J.*, N. S. vol. i. p. 385.

² *I. A.*, No. 21, p. 365.

to have a malarial origin. An East Indian woman, aged 24, was attacked in November 1866. Similar attacks had occurred in the Octobers of 1864 and 1865, when she had remained speechless for twenty days. For two nights previous to her admission, she had been sleeping on the top of her house, and had awakened feverish and speechless. Her countenance and gestures showed that she was perfectly intelligent; but there was complete aphasia. When a spoon was held before her, she could not name it, but expressed its use by gesture. When desired to write the word, she, at first, appeared quite unequal to the task. At last, after a prolonged contemplation, she began briskly to write the word which, it seemed, had dawned upon her; still she could not get beyond S. Eventually, after further desperate attempts to express the rest of the word, she succeeded; and very hurriedly wrote down, after the S, poon. She hurried over it, fearful, apparently, of again forgetting it. She knew her own and her husband's children's names, but appeared to have quite forgotten the surname. The attack lasted seven days, when, attempting to vomit, she called out to the nurse. On the following day, she conversed freely in English, Hindustani, and Bengali. She remained perfectly well for two days, and then had an attack of quotidian intermittent fever, with severe headache across the temples, which remained for some time after the fever had left her. Dr. Francis brought this case before the Bengal Branch of the British Medical Association, where the probability of the malarial origin of the attack was generally admitted.

Mr. G. D. McReddie¹ had a case which, although far less serious, resembled that of S., described above. A Brahmin, aged 45, being engaged in a riot, received a latti (bamboo staff) blow. There was only a slight contused wound on the left and anterior surface of the scalp. He was quite sensible, and his limbs were not paralysed; but he had lost the power of speech, he could not utter any articulate sound. On about the eighth day after the accident, he uttered the words 'Ram, Ram, Ram!' (a religious ejaculation) pretty plainly, but he failed to express any other word. After two months, he was quite well in other respects, and was set to some light labour. Two months and seventeen days after the receipt of the injury, 'he had regained, to a considerable degree, the power of speech, but his utterance was not quite distinct. He seemed not to have sufficient control over the movement of the tongue to regulate its action suitably for clear expression. He spoke as if drink had caused a temporary impediment in his speech.'

There is a case by Mr. May² of a sergeant, who had a large abscess occupying the whole of the anterior lobe of the left hemisphere. It involved the corpus callosum, and (doubtless latterly)

¹ *I. M. G.*, vol. iii. p. 131.

² *I. M. J.*, vol. ii. p. 128.

passed through it into the left ventricle. Six days before his death, he was found wandering about the barracks, speechless. On the first day he was twice bled to twenty ounces, had sixty leeches in two applications, and was purged by jalap, castor oil, and croton oil. On the following day, he was 'unable to speak distinctly.' On the third day, he did not speak, only nodding his head when questioned. On the fourth, there were two or three slight convulsions. On the fifth, and up to the time of his death on the sixth day, there were tonic contraction and violent convulsion of the *left* limbs.

Dr. Chuckerbutty had in his ward a very bright and intelligent-looking English tailor, approaching middle age, free from paralysis, whose speech was reduced to the word 'Yes!' He would listen with the closest attention to every question, and appeared to be quite elated whenever his word became available. Thus, when asked 'Have you a headache?' he would look 'knowing,' but remain silent; but, when one said 'I think that you have lost your headache,' he would jerk out a 'Yes!' and smile. I lost sight of the poor fellow—I believe that his friends removed him.

Sir Joseph Fayrer's case¹ of 'aphasia and death resulting from softening of the left anterior cerebral lobe and cerebellum, due to atheromatous degeneration and embolism of the cerebral arteries,' affords a most interesting study. The patient, aged about 42, was an officer of naturally great intellectual powers, much developed by scientific and literary study. He had three sudden attacks of apoplectic character, after each of which there was more or less complete impairment of the innervation of the right side. These covered about three years. He appeared to recover completely from the first, except that there remained an indistinctness in the articulation of certain words, and an unnatural rapidity of utterance. After the second, he seemed perfectly to comprehend any question that might be put to him, and attempted to reply; but the first three or four words had barely found utterance before he lapsed into the most incoherent and purposeless jargon, which appeared to indicate not only that the memory of words was lost, but that ideas, in the wildest and most incoherent jumble, supervened on the forgotten sounds. His attempts at writing were as incoherent as his speech.

Dr. Ewart² had an East Indian patient, aged 41, who had fallen down and become insensible on board ship on the night before admission. He had then rapidly become hemiplegic on the right side. When admitted, he had so far regained consciousness as to notice what was going on around him, but he remained quite speechless, and lay on his back at full stretch. He could raise the right arm and leg partially and slowly. He yawned frequently. He made

¹ *Op. cit.* p. 616.

² *I. A.*, No. 21, p. 355.

repeated attempts to speak, and wept bitterly when he found himself unable to articulate a single intelligible syllable. On the following day, the power of the right arm was entirely lost. On the sixth day, he became unconscious. On the eighth, consciousness partially returned. He remained for a month subsequently in Dr. Ewart's colleague's ward, and, being able to sit up, was discharged at his own wish. Early in the following month, he was placed under Dr. Ewart's care. His condition was that of ordinary partial right hemiplegia, his memory was most defective, and the faintly articulate language was almost extinguished. He could not remember his place of residence; but, when it was mentioned, he nodded assent and said 'Eh, sa,' for 'yes, sir.' When told to 'write down everything he knew about his illness,' he went on writing 'write down everything you know about your illness' over and over again, apparently without dreaming of the object of the injunction. When the report closed, four months and a week from the date of the fit, his general health had much improved, he walked about with ease, and was regaining power in the right arm; but, although there was improvement, the power of articulation remained very imperfect. With the exception of 'yes' and 'no' and his own name, there were few words to which he could give intelligible expression.

A patient of Dr. William Palmer's,¹ a French sailor, aged 45, had, after suffering much with illness and pain in the side on board ship, become suddenly speechless. Three days subsequently, there was not any paralysis, the pupils were slightly contracted, but were both active and equal. On admission, the expression of his countenance was rather dejected, stupid, and apathetic. When addressed, however, his whole face brightened up and beamed with intelligence; but, as soon as he attempted to frame the required answer, his expression became first confused, afterwards troubled, and then his eyes would close and his head fall back exhausted by the fruitless effort. After being allowed to rest awhile, he was asked whether he could write. He again nodded assent, but, when the pen was handed to him, he made no effort to do so, except to reach his hand towards it, when he again sank back exhausted and helpless. He could produce vocal and articulate sounds, but was utterly unable to form the simplest word. He died in a week with abscess of the liver. On a fold of brain, situated in the angle formed by the convolution of the *right* fissure of Sylvius, and then of the longitudinal fissure (Quain), immediately in front of the middle lobe, a circular spot of grey matter, about five-eighths of an inch in diameter, was softened. All the surrounding parts of the brain were quite firm and solid, and there was no appearance of arterial disease.

Not long before I left India, I had under my care for several weeks

¹ *I. M. G.*, vol. i. p. 177.

an oldish sailor man with secondary syphilis. He never spoke, but sat up in his bed reading cheap novels apparently with great attention. On coming to the foot of the second page, he tore the leaf out, and so regularly until he had 'finished' the volume. On examination *post-mortem*, I found a gumma, as large as a moderately-sized walnut, centrally situated in the *right* anterior cerebral lobe. The size of this mass was so considerable as to render it likely that its presence on the right side caused pressure on the left anterior lobe. Still, the most careful examination failed to discover any appearance of lesion on the left side. Although it was clear, during life, that this poor fellow's silence was true aphasia, I believed that his practice of tearing up books was due to ill-nature amounting to monomania. I think that I wronged him:—the following case appears to throw a partial light upon his condition. In the fifth series of 'Notes and Queries,' Vol. XI., p. 201, is an account, by his half-brother, of a very accomplished scholar, Mr. Hyman, Senior Fellow of Wadham College, Oxford, a man remarkable for the great retentiveness of his memory. In notices of his death in the journals, it was stated that 'he would tear out the pages of a book as he read it.' His kinsman confirmed this statement, saying, 'He himself, gave me, on more than one occasion, an explanation of his curious habit of tearing up his books as he read them, sometimes page by page, sometimes in handfuls. . . . The habit was, in fact, a bit of eccentricity, not entirely clear of something very like delusion.' His death appears to have occurred late in 1878, at about the age of 65. In 1857, twenty-one years before his decease, he gave his brother a book, saying, with a smile, 'You had better take it, *I shall only tear it up.*' Consequently, it is improbable that his mischievous habit was due to any organic cerebral disease.

Delirium Tremens.

Surgeon-General Hugh Macpherson shows that, in the eight years ending 1853-54, there were, in the European army of the Bengal Presidency, 2,448 admissions for delirium tremens, or $1\frac{1}{2}$ per cent. of strength. The deaths to treated were $6\frac{2}{3}$ per cent. Among infantry in the United Kingdom, the proportion of the former was only $\frac{1}{11}$ per cent., but of the latter it was $17\frac{2}{3}$. The returns show that, in the year 1880, the admissions of European soldiers, in all India, for delirium tremens were 232, being 3.9 per 1,000 of strength. The deaths were at the rate of 9.05 per cent. of treated. In 1881, there were 229 admissions, or 3.9 per 1,000 of strength. Deaths per cent. to treated, 4.37. In 1882, the number of admissions was 247—4.3 per 1,000 of strength. The deaths to treated were 1.21 per cent. There was not one admission among the soldiers' wives during the three years.

The main points requiring attention in this disease, as it occurs in

India, are that convulsion and epilepsy are, in my experience, more frequently present than they are in England, and that great heat causes predisposition to attacks, and not infrequently determines a fatal result.

I had just written this sentence, founded on many years' Indian observation, when I came upon the following remark, made by my friend Dr. John Macpherson¹ nearly thirty years ago: 'There is a presumption that heat favours the production of delirium tremens, and a still stronger one that it increases the mortality by it.'

In the early part of my career, the main object in the treatment of delirium tremens was to force sleep by large doses of opium, and to 'support' the victim by a very free supply of his wonted stimulant. Some years before I went to India, I witnessed the death, in a public institution, of an athletic young man, the subject of this disease, by opium poisoning. No one who stood about that death-bed denied that the poor fellow had been fatally overdosed. This and a salutary change in medical opinion, regarding the ætiology of the disease,² prevented me from administering opium, under any circumstances, in delirium tremens. The leading indications of treatment in my wards were to eschew opium and brandy, to purge the patient rather freely on admission, to keep him perfectly quiet, and as cool as possible in a shaded room, to nourish him throughout the attack, and to give him quinine in convalescence. I gave chloric ether in moderate doses, and when the patient begged pertinaciously for brandy, I let him have half-ounce in iced soda-water as a refreshing placebo. In exceptional cases, I solicited sleep by a small dose of hyoscyamus, but often found this unnecessary. These measures represented the practice of the majority of my brother officers for many years before I left India. They are admirably set forth³ by my friend Dr. Cayley, who gives the chief indications of treatment as being 'First, to prevent any more of the poison being introduced into the system; Second, to eliminate what is

¹ 'Statistics of Delirium Tremens,' *I. A.*, No. 5, p. 18.

² Morehead wrote (p. 631): 'Dr. Stovell has usually reduced the quantity of tincture of opium to ten minims, and in the expediency of this modification I am disposed to concur.' Further on, he cites Dr. Leith, who, writing at the General Hospital, Bombay, in 1857-58, said: 'The treatment of delirium tremens during the year has been chiefly expectant, and in the uncomplicated cases that have been under my own immediate care, no medicine whatever has been given. The patient is secluded, and kept as quiet as the present imperfect hospital accommodation will allow; and where there is any heat of head the cold affusion is used, and sometimes along with it the hot pediluvium. In many cases, however, even these remedies are not employed. Attention is always paid to alimentation, and strong broth is given at regular intervals. I had followed this plan for many years in the cases that, from time to time, came under my care, and now that I have had trial of it in a more abundant field of observation, I continue to be satisfied with it.' The mortality was 8 per cent.

³ *I. M. G.*, vol. i. p. 145.

already present as quickly as possible; and Third, to support the bodily powers, and keep the system in as favourable a condition as possible until the effects of the poison have passed off.'

I observed that, among my delirium tremens patients in India, a very large proportion were exceptionally powerful men of naturally fine constitution. One of them, who still occupied a responsible position, said to me, when at his best—'Delirium tremens is a mere nothing after the fourth or fifth attack.' But he added, 'There were a dozen of us D. T. subjects when I was in England a few years ago, but I am the only one who has not gone under!' I have already said that, amenable as the majority of our patients usually were to treatment, I never knew one with this disease who outlived the night following a heat-stroke day. In Calcutta, we always admitted the dead drunk to hospital, knowing that they would probably die in the police-cell.

The majority of young medical men, on going to India, have learnt that drunkards, in hospitals, generally lie unblushingly when questioned upon their habits; but, unless warned, they may not be prepared for the resolute pertinacity with which dipsomaniac '*gentlemen*' often maintain that they are patterns of sobriety. A scene like the following has occurred to me again and again. A person high in office, with whom I had been slightly acquainted, brought his brother to me saying that he was a man of independent means who had made a pleasure tour through India, during which he had suffered from ailments, headache, irritability of stomach, nausea, debility, &c., &c., which none of the medical men whom he had consulted had quite succeeded in making out. Here I received some papers, which I kept under my hand. His aspect revealed his story. I said, 'Tell me frankly—excess has brought you into this condition?' 'Certainly not!' his brother replied to my look of inquiry, 'Most certainly not!' 'Upon your honour?' Both, 'Upon my honour.' Then I looked at the official brother, thinking, 'I have a good mind to report you to Sir—as a deliberate liar!' I then turned over the papers—notes written by three up-country medical men—and said, 'Two of your three doctors say expressly what I have said. You must leave for home by the next steamer; and if, in the meantime, you do not act strictly upon my advice, you may not live to get on board.'¹ Here, as usually happens, the truth soon comes out. 'Well, I now and then feel so weak that I am obliged to take a quarter of a glass, &c., &c. ;' and, not infrequently, his medical advisers, holding the views of the late Dr. Todd, had also been 'so weak' as to allow the practice, and even to favour it.

I have met with illustrations of the fact that there are many unfortunate persons in tropical climates, especially those who have sustained injuries to the head or heat-stroke, to whom *total abstinence*

¹ I did not always succeed in pleasing my few aristocratic patients.

is the only measure by which respectability and health can be maintained. At one of my civil stations, a neighbouring brother officer (forty miles off) had suffered from delirium tremens. He took good advice, remained at T—— for ten years, abstained, and was well thought of. Being ordered away to Burmah, he had to join a mess, and was cashiered for drunkenness in a few weeks. I have seen many of these truly pitiable cases.

I have already spoken of the tendency to acute mania, especially in young full-blooded soldiers who are intemperate in India. The following observation, by Inspector-General John Macpherson, is, like all that he has written, full of practical truth : ‘ For the first three years when I was with European soldiers, I thought I could always cure the disease ’ (D. T.). ‘ After a period, some cases of sailors in Howrah staggered me ; and, when I came to the pensioners and invalids of the Calcutta General Hospital, I found that many cases were beyond all treatment.’ Here, doubtless, organic disease was much advanced.

I perhaps need scarcely urge the extreme importance of great tenderness in treating these patients when their suffering and fear render them violent, especially in the exhausting heat of a tropical climate. One morning, at Howrah, I was called to a very powerful-looking railway man, who, I was told, was a superior person who had left a family in England. On reaching the house, I found that, having been attacked with delirium tremens, he had escaped, and was rushing about all over the station, endeavouring to conceal himself in the gardens. I had started with others in pursuit of him, when a party who had captured him met us near the landing-place. Having no proper means for securing him, I put him on board a boat with several of his friends, and sent him to the Medical College Hospital. He was drenched in perspiration, pallid, and in abject terror, crying that I was sending him to be murdered. He died at the hospital that night.

Unquestionably, no class of gentlemen are more temperate than Indian officers generally are. The following case of acute alcoholic mania occurred in one who doubtless had a faulty brain, but was not habitually intemperate. Captain —— was in command of our small detachment of sepoys at —— . In rather hot weather, he went into the district upon a shooting expedition. Having met with a great deal of game, he was much excited, and it was noticed that he drank sherry with very unusual freedom. One evening, I received a note from his wife, which was literally to this effect : ‘ Captain —— has come back quite mad. He has his hog-spear, and declares that he will kill the first who approaches. Come to him immediately ! ’ Arming myself with two powders, I started, and, seeing his bungalow, lighted like a lantern, all the way, had time to reflect that this was a serious duty. As I approached, a blaze of light revealed him seated on a table surrounded by trophies of the chase, and holding his spear in one hand

and a foaming glass of beer in the other. Much to my relief, he appeared delighted to see me. Having received a glowing description of his sport, I said, 'I think, Mrs. —, that the Captain has had a hard day's work, and that all he needs is a glass of beer and a good night's rest.' A fresh bottle of Bass's was placed on the table, which was already crowded with bottles. I said that I would give it a fine head, and turning my back, frothed it up in a tall glass upon a drachm of ipecacuanha powder. This afforded great refreshment, and, another similarly medicated goblet having been accepted, I advised my commanding officer to go to bed. In the morning, I found him well and tranquil, complaining only of having been rather sick in the night.

Tetanus.

A description of the symptoms of this disease, which, in India, nearly resemble those observed in England, is not needful here. I shall confine myself to a few remarks upon points with which I became acquainted after I went to India.

Mr. Holmes has never spoken to anyone who has seen an example of either emprosthotonos or pleurosthotonos. I never saw or heard of a case of either in India, unless the following may be accepted as such.¹ 'Was transferred' [to the General Hospital, Madras] 'from the Eye Infirmary, where he had been under treatment for an injury to the eye caused by a spent rocket falling on it a month previously. Tetanus supervened. On admission, he was speechless; the muscles of mastication and deglutition were rigid, as well as the occipito-frontalis, (frontal portion) and the muscles of the front of the chest and abdomen. He was treated by chloral and bromide of potassium per rectum and nutritive enemata. But he sank and died of exhaustion on the third day after admission.' Dr. Leith's tables give a fatal case of emprosthotonos as occurring in Bombay in 1856.

Infantile Tetanus is prevalent in Calcutta. My friend, Dr. Kenneth Macleod, tells me that recent statistics have shown that it is extremely common there both in natives and Europeans. So I find² that Dr. Waddell mentions, in his report as chemical examiner for 1884-85, that the Government noticed that, during 1884, the Calcutta mortality returns gave 1,204 deaths from tetanus, the larger bulk of which occurred in children and infants; and it is suggested as worth considering whether many of these cases may not be due to strychnia, '*kuchilla*' poisoning, when it is known that nux vomica bark has been proved, on several occasions, to have been substituted for or mixed with '*kurchi*' bark (*Wrightia anti-dysenterica*) which is extensively used by the people for infantile ailments. In a valuable paper on the statistics of

¹ *Report of Civil Hospitals and Dispensaries, Madras, for 1883, p. 64.*

² *I. M. G. for 1886, p. 52.*

tetanus in the Calcutta Medical College Hospital,¹ Mr. J. R. Wallace observed that, among children and infants born there, trismus nascentium occurs more frequently in the children of Europeans than in those of natives, although the number of native births in the house exceeds that of Europeans. Out of 34 cases, 21 were Europeans, and this observation is all the more striking when we consider the comparatively small proportion the European element bears to the vast native population of Bengal. He found that infantile tetanus is written about as being of the idiopathic variety. If, however, it is held that the source of irritation lies in abnormal changes in the ligatured and divided cord, then it is truly traumatic. There is no doubt that, in many instances of Mohammedan and Jewish children in India, the disease is traumatic when the umbilicus is not affected. There, the practice is to circumcise the infant on the eighth day; and, in many instances, tetanic symptoms follow. He found that, in nine of the infants affected, the penis was the focus of irritation; and that, in two cases, the application of anodyne poultices was followed by recovery. Of the 34 cases of infantile tetanus, 29 were fatal. Baboo Lall Madhub Mookurjee considered that much of the tetanus of native children was due to the treatment of the navel. The cord is roughly divided with a piece of sharpened bamboo. Waring says that the Burmese midwives also divide the cord with a piece of bamboo split in the middle. Dr. Chambers was of opinion that infantile tetanus is due to bad hygiene.

It is noted, in the Madras Medico-Topographical Reports,² that, among the Burmese, in the cold season, infants are subject to trismus, which carries off many. Mr. S. Rogers, of the Madras Presidency, noticed, in his remarks on the case of a European female infant, which he treated successfully with the tincture of cannabis Indica, that trismus nascentium is a disease of very common occurrence in India, and, as far as he could ascertain, it had been invariably fatal.³ At the Ramasawmy Hospital, Madras, in 1883, four out of the thirty-one children who were born and died there succumbed to tetanus. There were not any cases at the Government Lying-in Hospital that year. In his remarks on tetanus⁴ Dr. Waring notices that trismus nascentium does not appear to be a frequent or, at any rate, a fatal disease in Bombay, where tetanus is very common in adults and women in child-bed. Dr. Leith's Bombay tables give only nine fatal cases, in infants, in six years.

Dr. D. B. Smith reported⁵ the *post-mortem* examination in the case of a (European?) infant at Mussoorie, in whom death (which, he

¹ Read at a meeting of the Calcutta Medical Society, December 1880.

² *Tenasserim Provinces*, p. 176.

³ *Madras Quarterly Medical Journal*, vol. iii. p. 65.

⁴ *I. A.*, No. 6 p. 505.

⁵ *I. M. G.*, vol. ii. p. 146.

remarks, is 'the rule in such cases') took place within twenty-four hours of the accession of the attack. In the spine, there was no increased vascularity, no effusion of blood or serum about the cord or its membranes. The meninges of the brain were much congested; the venous sinuses and meningeal veins gorged with blood. On the upper part of the convexity of the left hemisphere, was a layer of extravasated blood in the arachnoid cavity. There was a little serous effusion; the substance of the brain was softer than normal.

When, as not unfrequently happens in Lower Bengal, a native woman is brought to hospital with tetanus, we always have to decide the question—*Is this a case of abortion by mechanical means?* A clue to the real cause will probably be given by the statement that the attack has been excited by cold bathing during menstruation—which is not a native practice—at least in Bengal Proper. We often find that abortion has taken place, and that the uterus has been severely injured by the forcible introduction of a *lal chitra* (*Plumbago rosea*) stalk (a vesicant of extreme virulence) about a span long. (For much information on this subject, see my 'Medical Jurisprudence for India,' p. 712 *et seq.*) I need hardly insist that great care must always be exercised in the discrimination of such cases. Dr. Roberts, of Madras, has lately¹ remarked on the frequency of tetanus among native women who bathe in cold water on the third day after menstruation. That tetanus often follows natural delivery is shown in the remarks on Puerperal Tetanus. Mr. B. Dossabhoj reported² the case of a Parsee female, *ætat.* 26, the mother of four children, who aborted, apparently spontaneously, in the third month. On the seventh day, 'difficulty in opening the jaw to the full extent' was complained of. There were no further tetanic symptoms till five days later, when the abdomen was hard and tympanitic; without stiffness of the muscles except those of the jaw. She remained thus for twelve more days, when spasmodic contraction of the back and lower extremities came on, and there was increased stiffness of the muscles of the neck and abdomen. She recovered in about a month.

Remarks upon *Puerperal Tetanus* in India will be found in the section on the *Diseases of the Puerperal State*.

I observed a singular alliance—or at least coincidence—between *Muscular Rheumatism* and *Tetanus* in Lower Bengal. For many years past, I have been liable to wake, on a rainy gusty morning, with severe rheumatic pain in the muscles at the back of the neck. Movement is so painful that, at first, it is difficult to localise the affection; but, in a little time, I find that it is unilateral. This satisfies me; because, if it affected both sides, it would be trismus, or something very like it. On such mornings, the slightest attempt to stretch a

¹ *I. M. G.* for November 1885.

² *Bombay Med. and Phys. Trans* for 1853-4, p. 340.

lower limb causes most agonising cramp in the muscles of the calf. For some years before I left India, going to hospital with such a pain in the neck, my first question to my house physician was: 'Have we taken in a case of tetanus? if not, ask the house surgeons if they have admitted any, or whether any of their cases have been attacked.' The reply was repeatedly in the affirmative. Notoriously, stormy and damp weather excites tetanus. My father, who was surgeon of a line-of-battle ship at Trafalgar, and had fifty wounded and a great number of amputations, described the prevalence of tetanus after the great storm which followed the action, and in Gibraltar Hospital, among men with huge splinter-wounds, as being hideous, even to a surgeon.

Baron Larrey ascribed traumatic tetanus to moisture and sudden changes of temperature. He observed that the wounded men at the battle of the Pyramids, who were attacked with tetanus, were evidently the victims of the moisture and coldness of the night.

On the other hand, a Peninsular army surgeon told me that a soldier with traumatic tetanus, having been hastily stretched on an uncovered baggage waggon and so being carried on, much exposed throughout a clear cold night, was found to be free from tetanus in the morning.

Still again, Mr. Branfoot¹ has lately reported the case of a man who drove a long way in a dog-cart, his neck being exposed to a damp wind which gave him great discomfort. He was seized with tetanus, and died within twenty-four hours. At the same meeting, Mr. Drake-Brockman said that two ladies got tetanus from exposure to draughts at concerts. One of them was affected with tape-worm.

Everyone who has practised in India could probably cite cases of tetanus, from his own experience, in confirmation of Mr. Poland's observation² that 'there is scarcely a single lesion which may be said to be exempt from its attack, from the simple bruise or graze to the most severe compound fracture.' Many years ago, at Guy's, I watched the fatal case of a stalwart gardener in whom there was a small clean ulcer on the back of the hand. The presence of lumbrici in the intestines is probably a cause of tetanus in natives (*vide* Waring, *antea*, p. 31). They have been noticed in many fatal cases, but their occurrence is so general that it is difficult to prove that they have been the source of irritation. Assistant-Surgeon Cheytun Shah reported³ a case, in which recovery took place, where the causes of irritation were said to be thread-worms and otorrhœa. Since the publication of the above remarks, Surgeon-Major Rattan has observed⁴ that, in a great major-

¹ 'Report of the South Indian Branch of the British Medical Association,' *I. M. G.*, November 1885.

² Cited in the *M. T.*, vol. ii. for 1883, p. 603.

³ *I. M. G.* for October 1874.

⁴ 'Report of the South Indian Branch of the British Medical Association,' cited above.

city of cases of tetanus, a dose of santonin will dislodge round-worms. All of the patients lately treated for tetanus at the Madras General Hospital passed round-worms. In a young man for whom Mr. C. Macnamara ligatured bleeding piles, tetanus set in on the third day, and proved fatal in forty-eight hours.¹ Recently, Dr. Snamenski,² noticing the fact that ligature of external hæmorrhoids is always accompanied with excruciating pain, not to be relieved by narcotics, while that of internal piles is painless, suggests that this is probably the reason why trismus and tetanus so often follow the tying of external piles. The author adds two cases to many which he finds on record. In one of these, trismus and death followed the operation on the sixth day; while, in the other, most violent pains occurred, but, eventually, recovery took place. In thirty-nine cases of ligature of internal piles, neither pain nor complication took place. He finds, by experiment, that, in dogs, as in man, the mucous membrane of the rectum, one-and-a-half centimetres above the anus, is quite insensitive, while the last centimetre and the skin about the anus possess an extraordinary hyper-sensitive-ness. He thus confirms the researches of Fellner, who finds that the skin near the anus and the rectal mucous membrane for about a centimetre above the anus is provided with sensation by the sensory *ner. pudend. comm.*, while the mucous membrane of the rectum, above this point, receives only fibres of the non-sensory middle hæmorrhoidal nerves.

Several deaths from tetanus, due to unusual causes, appear in Dr. Leith's 'Deaths in Bombay.' 'From piercing of concha of ear, to cure hydrocele;' 'from blister applied;' 'sloughing of penis from constriction of iron ring;' 'rat-bite of knee;' 'bed sores;' 'serofulous ulcer of neck;' 'ulcer in groin.' Mr. P. Cullen³ treated a Hindoo, *æt.* 20, in whom the outward lesion was an open syphilitic bubo. Death occurred in about six days. A poor European man, long known in Calcutta as 'The Hero,' came to the house surgeon of my hospital and showed him a small ulcer on the shin. Upon having it dressed, he went away. We heard, very shortly afterwards, that he had died of tetanus. The house surgeon then recollected that the Hero had never uttered a word, although he was usually very talkative. As the sore appeared to speak for itself and as the man was very eccentric and apt to give trouble, his real condition passed unrecognised.

Dr. McKenzie recorded, in the 'Proceedings of the Hyderabad Medical Society' a case in which tetanus was caused by the irritation of a guinea-worm. A similar case is mentioned in the report on Coimbatore, p. 61 of the Madras Topographical Reports. No less than

¹ *I. M. G.*, vol. viii. p. 295.

² *Moscow Zeitschrift*, No. 35, 1884, and *M. T.*, vol. ii. for the same year, p. 768.

³ *I. M. G.*, vol. i. p. 177.

thirty-eight fatal cases of tetanus, attributed to guinea-worm, appear to have occurred in the City of Bombay in six years ending 1859.¹ Eighteen of these were registered in 1859. In two of these cases, the progress of tetanus was very rapid. Tetanus occurred in two soldiers, at Mussoorie, in whom the hypodermic injection of a solution of quinine was employed. A case of death from tetanus, occasioned by the hypodermic injection of fifteen grains of bromide of quinine in diluted alcohol, occurred at Iowa, U.S.²

In 1883, a young Hindoo had a hydrocele of four years' standing tapped, at the out-patients' department of the General Hospital, Madras. It was injected with thirty minims each of carbolic acid and glycerine. Severe inflammation was followed by tetanus. A strong spasm terminated his life on the morning after admission. Another hydrocele case is noted by Ballingall.³

Although its prevalence is greatest in hot and malarial climates, I am not aware that any observer, with the exception of my friend, Dr. Robert Bird, now of Cobham, Virginia, has noticed the association of tetanus with paludal fever.⁴ A son of a native gentleman at Bishop's College (a malarial locality of which I had medical charge for several years), after suffering much from ague and fever, was attacked with tetanus in the rainy season of 1861. This disease continued, in a less or greater degree, for ten days, when it suddenly left him on the supervision of high fever without ague.

It is often very hard to determine whether a case of tetanus is idiopathic or traumatic. (See the remarks above on Infantile Tetanus, p. 416.) I experienced this difficulty in the case of a little native school-boy at Howrah. Running out of afternoon school, he and his companions scudded over the plain until they came to a culvert a few feet deep. He fell in, but quickly scrambled out and ran home with the others. That night, he was fatally attacked with tetanus. There was not even the very slightest trace of outward injury. We were not allowed to examine the body *post-mortem*.

A closely parallel case appears to have occurred, in 1883, at Guy's Hospital.⁵ A boy, aged 9 years, died from tetanus two days after a fall from a ladder. Mr. Dendy, the house surgeon, said that he had been unable to trace the least scratch or wound, or even a sign that the deceased had had a fall, except that pressure about the neck appeared to give him pain. Nor did the autopsy show any injury, either local, or to the spinal cord, or elsewhere.

Soon after I arrived in India, I had a case in which I am unable

¹ Dr. A. H. Leith's *Deaths in Bombay*.

² Noticed in the *Lancet*, 1878.

³ *Military Surgery*, p. 255.

⁴ *I. M. G.*, vol. i. p. 57: 'Malaria and Some of its Effects.'

⁵ *M. T.*, vol. ii. for 1883, p. 603.

to say positively whether the tetanus was traumatic or idiopathic. At Purulia, I was breakfasting with Captain George Oakes, who told me that his khidmutgar (table-servant), who was in attendance, had a diseased elbow. He appeared to be unable to work, but was evidently unwilling to allude to his ailment. I found that the radius and ulna of one of his arms had been much necrosed throughout the upper halves. This had evidently occurred in early youth, as the growth of the limb had been arrested. All the sinuses had closed except at one spot where it appeared likely that bone would become detached. The arm was now rather swollen and painful when moved. He was of delicate frame. I did not probe, and I was glad to recollect afterwards that I moved the limb with the utmost gentleness. I enjoined rest. Two days afterwards, he was attacked with tetanus. I attended him throughout the night, but all treatment was utterly unavailing. He died in about sixteen hours. The pain in his arm had increased considerably. I could not obtain sight of the diseased structures *post-mortem*.

I treated and reported¹ the case of an East Indian woman, *æt.* 44, in whom an attack of tetanus, lasting three weeks, was preceded and attended by idiopathic sciatica. At one time, the spasms were of such terrible violence, principally affecting the diaphragm and causing extreme anginous præcordial distress, that death appeared to be threatened.

I believe that the whole of these cases were, ætiologically, traumatic. In the boys, there were shock and concussion. It is probable that, in the man, a portion of dead bone was causing irritation. In the woman, there was distinct neuritis of the sciatic.

Duration of Tetanus.—Under the heading of *Spasm of the Glottis* (Diseases of the Respiratory System), I allude to the alleged case of a negro in Edinburgh, and to that of a little patient of Dr. Allan Webb's, who were considered to have been choked by the first spasm of tetanus. Mr. Wallace, of Calcutta, says that, in 212 fatal cases, the duration of tetanus was, within 2 days, 88 deaths; from 2 to 5 days, 41; from 5 to 10 days, 26; from 10 to 22 days, 23; above 22 days, 34. Out of 327 fatal cases cited by Mr. Poland, 79 died within 2 days; 104 in from 2 to 5 days; 90 from 5 to 10 days; 43, 10 to 22 days; 11 beyond the 22nd day. Instances of death up to the 39th day are recorded by Mr. Poland.² In India, both idiopathic and traumatic tetanus often last for a month or longer, with varying result. It appeared that, although the natives' sufferings during this prolonged ordeal were great, their lives were often protracted, because the clonic contractions of their muscles were less intense in them than in Europeans. I have made the like remark with regard to the spasms of cholera, p. 296.

I saw death occur from a sudden spasm in a muscular young native,

¹ *I. A.*, No. 4, p. 677.

² *System of Surgery* and *Holmes's Surgery*.

during amputation of the forearm, under chloroform, for tetanus. When their disease was far advanced, two of my idiopathic cases were attacked with cholera and recovered.

Statistics of Tetanus.—We learn from Macpherson's tables that, in the eight years ending 1853–54, there were, among European soldiers serving in Bengal, eight cases of tetanus with three deaths, or 37 per cent. In the latter four of those years, two women were attacked, no children.

In 1880, there were four fatal cases of this disease in the European army throughout India. Among the sepoys, there were 7 cases, of which 6 were fatal. In the jail population, there were 21 cases, 19 fatal. In 1881, the European soldiers had no loss from the disease; of their children, 2 were admitted, one of whom died. Among the sepoys, 7 were attacked, 3 of whom died. Of the prisoners, 18 were attacked, 15 fatally. In 1882, no European soldier died of tetanus; 8 sepoys were attacked, of whom only 1 survived. Among the prisoners, 11 cases occurred, with a fatal result in 9.

Dr. John Jackson, now of Brighton, had great experience of tetanus at the old Native Hospital, Calcutta, which admitted many injured lascars, and native grooms hurt by horses in the neighbouring stables. In 1854, he published a very important report,¹ which is founded upon 50 traumatic and 6 idiopathic cases admitted in five consecutive years. Contrary to what was then the general opinion, he found the traumatic form much more tractable than the idiopathic. In the traumatic variety, the percentage of deaths was 35; in the idiopathic, 50 per cent.

Dr. Peet published² a report in which he analysed 195 cases of tetanus which occurred in the Jamsetjee Jeejeebhoy Hospital, Bombay, between 1845 and 1851. Of these, 145 were fatal. Out of 75 admissions for the idiopathic form, there were 57 deaths. In 120 cases of traumatic tetanus, there were 87 deaths. Dr. Peet concluded that the idiopathic type is much more frequent in Bombay than in other parts of the world; that it is more severe and fatal than the traumatic species, and that it is often traceable to direct exposure in those seasons during which there are the greatest alternations of temperature. Morehead shows that, during the five years 1848–52, there occurred 1,716 deaths from tetanus in Bombay, or 2·5 per cent. of the total deaths. His chapter on this disease (xxix.) may be studied with the greatest advantage in conjunction with Dr. Jackson's and Dr. Peet's reports.

Dr. Waring³ gives statistical evidence of the fact that 'tetanus is' (or was when he wrote) 'an extremely fatal disease in Bombay, 1 in every 46 deaths being from this cause. A similar proportion of deaths is not

¹ *I. A.*, No. 1, p. 58, 'Notes on the Treatment of Tetanus.'

² *Transactions of the Medical and Physical Society of Bombay*, No. 1, N. S. for 1851–2.

³ 'Notes on Some of the Diseases of India,' *I. A.*, No. 6, pp. 501 and 512.

found in northern latitudes. Season, *per se*, appears to exercise little influence on these deaths from tetanus, a nearly equal proportion of deaths taking place in each of the four quarters.

Baboo G. C. Chatterjee published¹ the statistics of 132 cases of tetanus treated in the Calcutta Medical College Hospital during the four years 1861-64. Of these, 18 occurred in Christians, and 114 in natives. There were 96 traumatic and 36 idiopathic cases. The issue of 9 cases is not given.² There were 53 recoveries and 70 deaths. Of the traumatic cases, 37 recovered and 53 died; and, of the idiopathic, 16 recovered and 17 died.

In Mr. Wallace's paper, cited at the beginning of this chapter, we have an almost exhaustive statistical analysis of 280 cases of tetanus treated in the above-named hospital, during the ten years 1869-79. Besides these 280 cases, 23 others occurred in the surgical wards following operations of various kinds. All of these terminated fatally. Of the 280 cases, 159 were idiopathic, with 111 deaths; 121 were traumatic, with 101 deaths. There were 68 recoveries in 280 cases. Of these, 53 were among the idiopathic, and 15 among the traumatic. In the discussion which followed the reading of Mr. Wallace's paper, Dr. McLeod remarked that the comparatively low death-rate in traumatic tetanus is surprising. The doctrine taught in the text-books is that it is almost invariably fatal. (See Dr. Jackson's and Peet's observations at p. 422.)

In drawing up statistics of Indian tetanus, it is always needful to state whether puerperal and infantile cases have been taken as traumatic or idiopathic. In this respect, none of the above statistics, with the exception of Dr. John Jackson's, are perfectly clear. In Dr. Jackson's hospital there were no women or infants. Mr. Wallace accounts the puerperal cases as idiopathic; hence, in a large measure, the high mortality rate which he assigns to the idiopathic type. I regard them as traumatic.

Treatment of Tetanus.—Dr. O'Shaughnessy, now Sir William O'Shaughnessy-Brooke, was the first to draw attention to the value of *cannabis indica* as a remedial agent in tetanus and other diseases of the nervous system. His paper 'On the Preparations of the Indian Hemp or Gunjah (*Cannabis Indica*): their Effects on the Animal System in Health and their Utility in the Treatment of Tetanus and other Convulsive Diseases,'³ deserves the attention of every Indian practitioner. The late Dr. Chuckerbutty published, in 1868, a report on the treatment of tetanus by large doses of Indian hemp.⁴ I used and valued the drug in this disease.

¹ *I. M. G.*, vol. i. p. 3.

² These were probably removed by their 'friends'—an almost daily occurrence in Indian hospitals.

³ Published in 1839, *C. M. T.*, vol. viii. part 2, p. 42.

⁴ *I. A.*, No. 24, p. 187.

Baboo A. C. recommends¹ *smoking* of the Gunjah (cannabis) leaf. An attendant keeps a pipe ready near the patient, charged with, say, 15 grains of dried Gunjah leaves alone or mixed with twice as much of dried tobacco leaf mashed together, and watches the patient's movements. On the appearance of a clonic spasm, he is made to smoke the pipe until the leaves are burnt to ashes, on which the muscles instantly relax, the patient shuts his eyes and apparently falls asleep. This is carried on day and night uninterruptedly. Cure [recovery ?] takes place in from seven days to six weeks.

Sir Joseph Fayrer employed opium-smoking, hemp, chloroform, quinine, and a nutritious diet, free action of the bowels being maintained.² He attributes the ill-success of operative intervention to the fact that it has generally been employed too late, when the disease is thoroughly established. He suggests the advantage of resorting to amputation, even of a leg, on the earliest invasion of the symptoms. Amputation, he is quite aware, has often been practised, and with unfavourable results, but he is inclined to believe that it has not, as a general rule, been resorted to early enough to give it a fair chance of success. He adds that no one would recommend amputation of the limb, if the peccant nerve could be isolated and divided, as in the case of that leading from a finger or a toe. In a wound of the leg or arm, it is almost impossible to indicate the branch or trunk that is the conductor of mischief to the centre, and therefore, to secure its division, the whole limb must be removed. With regard to the cases which he reviewed, Mr. Wallace found that 'the practice in which surgical operations were performed for the relief of the sufferer has been attended with the most unfavourable results. In two cases, the sciatic nerve was stretched for tetanus, arising from wounds of the foot; in one instance, the median was subjected to the same operation for disease originating in a comminuted fracture of the thumb, and in neither of these cases did abatement of the spasms or other improvement in the patient's condition attend the procedure. Amputation of the hand in two cases, of the forearm in three, and of the leg in two, was resorted to without effect.'

Dr. John Jackson relied mainly upon the repeated inhalation of chloroform, added to the internal use of extract of hemp and aloes; and, in the latter stages, of quinine and generous diet. In speaking of Dr. Jackson's plan of treatment, Dr. Morehead observed: 'There is, I apprehend, very little difference in the principles of treatment respectively advocated by us; Dr. Jackson perhaps attaches more value to chloroform than I am disposed to accord to it.'

In Calcutta, Dr. F. Macnamara gave hydrate of chloral day and night; 30 out of 83 cases recovered. Sir Joseph Fayrer employed this drug.³

¹ *I. M. G.* for August, 1878.

² *Observations*, p. 179 *et seq.*

³ *Op. cit.* p. 185.

He considered that it might have had something to do with recovery in some of his cases; but, in others similar, he did not find it successful, except in giving euthanasia.

Dr. Cayley has shown that the statistics which he had collected from the records of the Mayo Hospital, Calcutta, and published in the 'Indian Annals of Medical Science,' do not furnish evidence in favour of the use of chloral. From 1871 to 1874, before chloral had come into use, 49 cases of traumatic and 56 of idiopathic tetanus had been treated, among which 23 and 26 deaths had occurred, which yielded percentages of 47 and 46. In 1875-76, when chloral was extensively used, the corresponding figures were: cases 28 and 23, deaths 14 and 14, or 50 and 64 per cent.

Professor Verneuil¹ reports great success with chloral in the treatment of traumatic tetanus, by packing in wadding and by inducing a kind of continuous coma which is to be prolonged *at least* fifteen days, if not more. Tracheotomy has furnished him with some remarkable recoveries where death from laryngismus has been threatened.

I need hardly say that we have no proof (save that afforded by a very few not quite indisputable cases in which cessation of spasm is said immediately to have followed amputation above the seat of lesion), that an attack of tetanus can be cut short by any mode of treatment. Our patient may die suddenly, by spasm of the respiratory muscles or cardiac apnoea at any moment, or he may sink by asthenia, slowly or rapidly tortured to death by muscular spasm, at any time up to six weeks. The main indications of treatment, consequently, are (1) to subdue spasm by means tending as little as possible to exhaust nervous power; and (2) to give the powers of life all possible support throughout the trial. Using hemp and quinine, and feeding the patient night and day, I surrounded the bed with screens, and poured chloroform very freely on the pillow, so as to keep the sufferer constantly in an atmosphere strongly charged with chloroform.

Being a surgeon's son, I, even in my school-boy days, listened intently to conversations on medical subjects. A venerable man, who had been a noted practitioner in the West Indies, said that he had been told of a slave who was treated in tetanus by having a deep incision made, on either side, down the whole length of the spinal column. These ghastly wounds were filled with boiling pitch, and, upon this, a straight board was fastened from nucha to sacrum!

More than once, on being called in to examine a malingering scholar, I have found that the narration of this case to the family doctor, taken a little aside, has at once dispelled a formidable array of anomalous nervous symptoms, as if by a charm.

¹ *Gazette des Hôpitaux* and *M. T.*, p. 360, vol. i. for 1885.

Rabies.

Macpherson found that, among European soldiers serving in Bengal in the eight years ending 1854, there were five cases of hydrophobia with three deaths. One soldier's wife was attacked and died.

The returns show that, in the three years 1880-82, there were 6 deaths of European soldiers; 5 admissions of sepoys, and 4 deaths; 2 deaths of soldiers' children, and 2 of native prisoners; in all 15 cases of hydrophobia. It is, however, to be borne in mind that those whose health statistics are given in the Sanitary Commissioner's returns are probably less exposed to hydrophobia than any other class of persons in India.

This malady is sufficiently common in India to enable—or rather to compel—nearly every man of experience to see cases; and, consequently, the Indian medical man rarely keeps a dog. Morehead treated ten cases. I had five. The bite of a jackal is especially dreaded, because its teeth are extremely sharp, and because this timid animal never attacks a man unless it is hydrophobic. I was standing in conversation with a gentleman at the door of a Calcutta house, when a rabid jackal rushed through between us, but, happily, did not snap. It was immediately killed by the grooms. The camp followers of a force of which Mr. Spilsbury¹ was in medical charge were attacked, while sleeping in the night, by a mad jackal, which bit some twelve or fourteen most severely. Most of the sufferers immediately applied for assistance. The bitten parts were instantly bathed and rubbed with hot water. This was done for a considerable time, so as to procure as great a flow of blood as possible, and the wounds were then freely cauterised with a stick of lunar caustic. The whole of those treated remained well up to the end of fifteen months. Three men, who were not treated, died, two in about six weeks, the other in ten months, with the most marked symptoms of hydrophobia. Mr. Spilsbury added that, some years previously, a very similar circumstance took place; and, as many of the people were bitten in parts where excision could not well be practised, he had recourse to the above treatment with the same happy results. I doubt the utility of the bathing. At Chittagong, two poor children were brought to me who had been miserably scored down their naked bodies by the teeth of a rabid jackal. I used the solid nitrate of silver very freely; I could not ascertain the result. Hydrophobia has been caused by the bite of a mungoose, a very fierce little animal which is often domesticated.²

I believe that, in its main features, hydrophobia is the same in all

¹ *I. M. J.*, N.S. 3, p. 224.

² *I. A.*, No. 11, p. 201, by M. S. Moodelly.

places ; but, in India as elsewhere, every case has, in its symptoms, a marked individuality of its own.

Seeing how frequently dog-bite occurs, how prone mankind are to hypochondriacal terror, and how intensely hydrophobia is dreaded, it is singular that *Hydrophobic Monomania* should be of rare occurrence. Mr. Austin Fitzgerald told me of a native doctor of his who appeared to have been long the subject of hydrophobic melancholia, being hopelessly impressed with the idea that the poison had existed for years in his system.

Dr. Campbell related¹ the odd case of a Nepaulese Brahmin, aged 18, who complained of pain in the throat on swallowing water, and was believed to be in the first stage of hydrophobia, having been bitten on the thigh by a mad dog about three months previously. A strong idea that the dog and its whelps were running up and down in his inside and barking in his belly was driven out by violent purgation and a course of mercury. This idea of the dogs was rather a popular than a personal delusion, the following notion being universally credited by the natives of Nepal : ‘ Shortly after the bite, the bitten person gets pups of the mad dog in his belly. At about three months, the young ones begin to move rapidly up and down the patient’s intestines, and, being mad like their progenitor, they bite and bark incessantly. This continues for some days, during which the young ones grow rapidly in size and strength, while their madness increases apace. This state of things lasts until they kill the unfortunate being thus so cruelly possessed of the canine litter.’² The Brahmin returned home, apparently quite well, in seven days, after which Dr. Campbell heard nothing further of him.

A very important paper on *Simulated Hydrophobia* was contributed by the late Dr. Bryden to the first volume of the ‘ Indian Medical Gazette.’³ Dr. Bryden had all the army statistics to refer to, and found that, especially during the very trying mutiny campaign of 1858–59, our unacclimatised European army afforded many cases in which the tendency to (1) insolation and (2) delirium tremens, or to a combination of both, was characterised by symptoms closely resembling those of hydrophobia. Reading the question by this light, we agree with Dr. Bryden in thinking that we perceive the true nature of the three following sets of cases—those in which it has been supposed

¹ *I. M. J.*, vol. i. p. 202.

²

About her middle round

A cry of hell hounds never ceasing barked,
With wide Cerberean mouths full loud, and rung
A hideous peal ; yet, when they list, would creep,
If aught disturbed their noise, into her womb,
And kennel there, yet there still barked and howled
Within unseen.

Milton’s *Sin*.

³ Page 57.

that true hydrophobia has been cured, those in which hydrophobia has occurred without inoculation, and those in which the bite and the symptoms are separated by an interval of years. Dr. Bryden cites the case of a young man, attached as an apprentice to the Allahabad General Hospital, who had been bitten three years previously. He was in the habit of saying that he should go mad, and, long before his last illness, had asked Dr. Wright whether he would die of hydrophobia. In the hot season of 1862, he died with exactly the symptoms of hydrophobia; but, as Dr. Bryden remarked, the development of this poison, latent in the system for three years, is a possibility which cannot be admitted.

Dr. Bryden's commentary was prompted by a very remarkable case observed¹ by Sir Joseph Fayrer, of a mechanical engineer, aged 23, of very excitable temperament, who, about thirty days previously, had been slightly bitten on the cheek by a pet spaniel. The weather was extremely hot, and he had been considerably exposed to the sun. Shortly before the accident, he had read a story in 'Chambers's Journal,' entitled, 'The Longest Month in my Life.' It was an account of a person, bitten by a dog, who was assured by a surgeon that he was not safe until thirty days had elapsed. This story seems to have made a great impression on his mind, and he had brooded deeply over it. 'He had no dread of fluids; he looked at, touched, and drank them without terror, but with occasional spasm; cold he did dread. Otherwise, the symptoms were those of rabies; and, although they were of far less intensity than is usual in this disease, they led up to death in about three days. The whole narrative well deserves the student's attention. There was no evidence that the dog was mad; no one suspected it; nothing had led anyone to suppose that it was so. It bit the patient as it were by accident, when he was playing with it. He unfortunately killed the dog directly afterwards.' Here we see, ineradicably implanted in the superstitious belief of educated Englishmen, a delusion as gross as the Nepaulese fancy cited above, and far more mischievous. I found, strongly fixed in the mind of a high Bengal civilian thirty-seven years ago, and in the opinions of an English gentleman's family not many months since, this irrational notion:—If you are bitten by a perfectly healthy dog, you will get hydrophobia, should it, at any future time, be bitten by a rabid animal!²

¹ *Observations*, p. 544.

² The following early notice of this superstition appears in Thomas Lupton's *A Thousand Notable Things*, 1660, p. 127. 'Whosoever is stricken or hurt by any venomous worm, or other thing, or else bitten with a mad dog, let them take heed diligently that the same thing that did hurt them see them not until they be perfectly whole. For the Hebrew Physitians say that the party hurt shall then dye, or else be in perill a fresh; yea, though they begin to wax whole when they see them. Anthonius Mizaldus.'

I can throw no useful light upon the TREATMENT of hydrophobia by drugs. Dr. Babington, cited by Aitken, gave 180 grains of solid opium in eleven hours without the least narcotic effect, or the slightest mitigation of the symptoms.

I plied one poor sufferer with morphia, in rapidly increasing doses, as long as he would swallow them. He took thirteen grains within a few hours. The drug produced no more appreciable effect in mitigating his hideous paroxysms than so many grains of chalk would have done. It will be seen that, in my case in which tracheotomy was performed (p. 431), morphia was given freely without any appreciable effect. In another extremely severe case, frequently repeated doses appeared to promote euthanasia; and, in a fourth patient, a hypodermic injection of morphia also seemed to have a like effect, but this was late in the disease.

Whenever the patient could be kept at all still, I surrounded him with an atmosphere of chloroform, with which the pillow and thick flannels placed about him were saturated. The Indian practice of giving small pieces of cucumber or melon now and then appeared useful in mitigating thirst.

Surgeon-Major Keegan, of Indore, has published¹ the case of a native who died of hydrophobia, in which it appeared certain that the application of a five, and afterwards of a ten per cent. solution of muriate of cocaine freely over the back of the fauces and upper part of the larynx gave very great relief to his patient by enabling him to drink freely. What of cocaine spray to the larynx?

In common with the whole profession, I await with great interest the fully developed result of M. Pasteur's operation.

In 1854, I published² a full review of the then current opinions upon Marshall Hall's recommendation that tracheotomy should be employed in epilepsy, convulsions, tetanus, and *hydrophobia*. I then cited the case of a medical officer of the Bombay Presidency³ who, as long previously as the year 1826, when suffering the agony of death from hydrophobia, '*declared himself patient to endure whatever we wished, and even suggested tracheotomy.*' This, so far as I can discover, appears to have been the first suggestion of the operation, elicited by the sufferings of one who was well able to perceive that the dreadfully convulsed and agitated state of the muscles of deglutition, and agonising sense of suffocation could only be relieved by the formation of an opening in the air-passage below the influence of muscular spasm. In that article, I cited and reviewed all that had been said by Dalziel, Herbert Mayo, and Marshall Hall in favour of tracheotomy in hydrophobia, and combated, with great deference, Sir Thomas

¹ *I. M. G.* for March 1885.

² *I. A.*, No 2, p 735.

³ Dr. R. H. Kennedy, *C. M. T.*, vol. ii. p. 383.

Watson's objections to this measure. Thenceforward, I resolved to put the validity of tracheotomy to the test in a case of hydrophobia.

In 1858, my friend, and now again my near neighbour, Brigade-Surgeon J. Barclay Scriven, performed laryngotomy with perfect success as regards laryngismus, general convulsions and horror of water, in a seaman, aged 19, at the Calcutta General Hospital, just as the patient was dying of laryngismus, in an attack which presented all the features of hydrophobia, save that evidence of the bite of a rabid animal was wanting. Full details of this very important case are given in No. 11 of the 'Indian Annals of Medical Science,' and in the 'Lancet' for April 23, 1859. Mr. Scriven wrote: 'About 8 P.M. on August 8, the patient was suddenly seized with a "feeling of weakness, wind at stomach, and difficulty in breathing," while on the staircase, which obliged him to hold on by the balusters. The apothecary was sent for and gave him a dose of peppermint, which was said to have relieved him temporarily. At 8.45 P.M., however, I received a note to the effect that a patient in the ward was complaining "of choking." I went over at once and found the man sitting up on the bed, labouring for breath, yet taking deep sighs and filling his lungs completely; he was in great distress, with small rapid pulse, perspiring profusely, crying out that he was dying for want of breath, complaining of pain in the throat, and requesting me to do something for him immediately. Presently, a decided spasm of the glottis came on, with crowing inspiration, like that of laryngismus stridulus in the child. I gave him a drink of tea, which he at first said would choke him, but which he afterwards took with apparent avidity. For a few seconds after this he had no crowing, though he still laboured for breath as at first. The spasm, however, soon returned with the crowing as before, and was again apparently relieved by a draught of water. This was repeated three or four times, but, finding the relief only momentary, I sent for a mustard emetic. He put it to his lips and drank some of it; then, by an apparently half-convulsive movement, he threw the remainder on the ground, and fell back upon his bed unable to breathe from spasm of the glottis. There was no crowing then, for the glottis was completely closed. I had my penknife ready, and plunged the small blade into the larynx, through the crico-thyroid membrane. The man was struggling so much at the time that I only succeeded in making a small opening the size of the blade, through which, however, the air whistled, and the relief instantly afforded was obvious to all present. He now began to talk very fast and loud, expressing excessive fear of death, and calling upon God to have mercy upon him, at the same time struggling so that about six men were required to hold him. By seizing opportunities, I gradually managed to enlarge the opening into the larynx by cutting through the cricoid cartilage, and to insert a quill through which he breathed freely. He continued to

talk in the same rapid way, and soon became decidedly incoherent.' 'It was impossible to quiet him or arrest his attention, but any unexpected movement made him start and talk the louder, as when I placed my hand upon the temples to feel the artery. I sent for a bheesty and poured a bag of water on his head, but it had little or no effect, except to increase distress and excitement, especially when the water ran into his mouth. I tried to give him some brandy and water and some small lumps of ice, but they did not seem to do good, and the greater part of both he managed to spit out.' On being removed to a quiet place, 'he struggled much less, but still continued talking in the same strain, though less loudly, occasionally spitting out with seeming difficulty some viscid saliva. His pulse was small and going 160 in the minute; skin hot and moist, great sensibility of surface, he started even when touched; the pupils were dilated and insensible. Respirations were principally thoracic; he had no spasmodic or convulsive action of muscles. At 11 P.M., he swallowed a little brandy and water that was dropped into his mouth. He continued in this state for about half an hour; then, without evident cause, became greatly excited, bawled so as to be heard all over the compound, and required to be held by several men, at whom he spat vigorously. Suddenly, it appears, he became sensible when spoken to by the ward-master, stood up to have his bed made, and afterwards lay down, took a little brandy and water, and went to sleep. He was awake at 4 A.M., when I went over to see him, was quite cool and rational, but complained of being very weak.' He recovered from the hydrophobic symptoms, but continued to be the subject of melancholia with a suicidal tendency. He was transferred to the lunatic asylum, and in five months was sent to England, 'not having lost the conviction that he had committed some heinous sin.'

The following case of hydrophobia, in which I opened the trachea, is drawn up from the notes of my house physician, Kalla Chand Holdar, and my own.

Reyda Chand Dutt, aged 20, Hindoo, a household servant, a native of Burdwan, a slight but tolerably muscular and healthy young man, was admitted to the Calcutta Medical College Hospital, at half-past one o'clock in the afternoon on April 29, 1863. The patient's uncle stated that, about two months previously, he was attacked in the road by a strange dog and severely bitten on the outer part of the left ankle, under the malleolus. The wound was very slow in healing, not being completely cicatrised until about a fortnight before his admission. He was not certain whether the dog was mad or not. He took many native remedies of reputed value as prophylactics of hydrophobia. Four days before admission, he suffered from a sense of general uneasiness, with some obscure pain in the back and limbs, and with slight heaviness of the head and distaste for food. On the day previous

to his admission, at about 10 o'clock A.M., he attempted to take his morning meal, but was prevented from swallowing by a choking sensation in the throat. With the occurrence of this symptom, the pain in the back began to increase, and that in the limbs changed from a dull to a gnawing character. During the night, the pain in the back gradually extended upwards to the neck, which became and continued somewhat stiff. At about seven o'clock on the evening previous to his admission, he succeeded in swallowing a few drops of water, but with great suffering. During that night, he slept very badly, suffered a good deal from pain in the back and limbs, and was attacked five or six times with spasms. At about eight o'clock on the morning of his admission, a sensation of cramp about the pharynx became troublesome and continued. The principal symptoms, on admission, were gnawing pains in the limbs and a dull pain in the back; stiffness about the neck and suffocative paroxysmal spasms in the muscles of the larynx and pharynx, pain in the thorax, and a sense of tightness below the ribs. When spasm commenced about the pharynx, a peculiar pain was experienced about the epigastrium. It was then evident that the whole of the muscles of respiration and deglutition were seriously involved. The spasms recurred every ten or twelve minutes; and, during their continuance, he suffered from much difficulty in breathing.¹ The breathing was much easier during the intervals. There was a peculiar wild and anxious expression of countenance. Forehead beaded with perspiration. Pulse small and quick, 90 in the minute. No urine or stool to-day. Is hungry and has great thirst, but cannot swallow anything, each attempt to swallow exciting suffocative spasms. Is not affected by *the sight* of liquids. Spasms are now and then excited by touching the body. Surface of natural temperature. There is no hawking or increased secretion of saliva; but he, from time to time, licks his lips as if they were sticky.

There is a raised callous cicatrix, two inches long, about an inch below the left outer malleolus. It is neither tender nor sensitive under pressure, nor is it the centre of any radiating pain.

Ordered half a grain of morphia, in powder, immediately, to be repeated in half an hour, and then to be continued every hour.

6 P.M.—The suffocative spasms have much increased in severity, respiration much more embarrassed. The surface of the body and limbs is bedewed with a cold sweat. Is becoming very irritable. Pulse small, quick and occasionally irregular. Continue the morphia.

As the evening advanced, all the worst symptoms increased in violence, and the patient's strength gradually failed under the progres-

¹ More stress is laid upon the *suffocative* character of the spasms in my very intelligent House Physician's notes of the case than in my own. Respiration was seriously embarrassed during the paroxysms, but it did not appear to me that death from suffocation was positively threatened.

sively augmenting severity of the spasmodic paroxysms. The pulse became smaller, slower, and more irregular; great pain in the neck was complained of. I now began to entertain the conviction that the operation of tracheotomy afforded the patient the only chance of valid relief. While watching the advancing severity of the spasms I made the following note:—

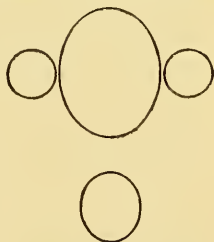
‘I am by no means certain that this patient is threatened with death by spasm of the glottis, considering that he has said repeatedly that he feels no uneasiness in that situation during the spasms, and that there is nothing very distinctly suffocative in the character of the spasms. His chief danger appears to be from asthenia. He has a quick, small, intermitting, and irregular pulse, and he has not taken food either yesterday or to-day. Still, must there not always be something suffocative in the character of such spasms as these? Will not an opening in the trachea enable him to swallow? Would it not be a great advantage, at least, to insure him against all possible risk of sudden asphyxia caused by closure of the glottis, or from gradual exhaustion aggravated and hastened by attacks impeding respiration, and, thereby, still further disturbing the brain and heart?’

At 10.15 o'clock at night, I performed, by candlelight, the operation of tracheotomy. The patient was persuaded to undergo it without much difficulty, and bore it exceedingly well. It was rapidly and easily done, and was not interrupted by any considerable spasm; the fullest sized curved trachea tube available was introduced without troublesome bleeding, although a small integumentary artery threw out a florid jet. The freest respiration through the tube was immediately established.

These impressions show the size of the orifices of the tube employed.

As had been anticipated, the relief produced by the operation became immediately evident. Although a spasm came on whenever ice was given to him, he took small lumps into his mouth with great avidity. *He also took several table-spoonful of cold water.* The spasms, however, increased in violence with every spoonful, his eyes protruding, his hands quivering, all the muscles of his neck working like cords, the air rushing out through the canula in sibilant blasts.

It was manifest that, previous to the operation, every attempt to drink created violent spasmodic action in three sets of muscles: (1) the pharyngeal, (2) the laryngeal, and (3) the diaphragm and thoracic muscles of respiration. Probably there was also (4) cardiac spasm. The operation immediately allayed and put a final stop to the injurious effects of contractile spasm in the glottideal muscles. Consequently, the patient was, at first, enabled to swallow liquids, subduing with



difficulty the resistance of the pharyngeal and respiratory muscles. The spasmodic action in these two sets of muscles, however, gradually obtained the ascendancy, and deglutition became more and more difficult at every draught. My observation of this patient's condition convinced me that, while tracheotomy in hydrophobia effectually prevents suffocation from spasm of the glottis, pharyngeal spasm persists, and it is still possible that death may be caused by spasm of the pharynx, thoracic muscles of respiration and diaphragm, and doubtless also of the heart. This, however, was not the issue of my patient's case.

As far as it went, the effect of the operation was decidedly good. It afforded him very considerable temporary relief, giving an interval of comparative ease from attacks of spasm, and enabling him to quench his thirst.

Half a grain of morphia was given after the operation (making $4\frac{1}{4}$ grains taken within about nine hours). This medicine was ordered to be continued, in quarter-grain doses, every hour.

At 11.45 o'clock, an hour after the operation, he was very quiet, and had been comparatively free from spontaneous spasms, having only been moderately attacked twice. He took a dose of morphia without embarrassment, *and swallowed six tablespoonsful of strong soup in succession*. The first spoonful was gulped down at once with great difficulty, overcoming strong spasm. In fact, now, as before the operation, spasm attacked him at every attempt to swallow liquids; but he was now able to contend with it, and to swallow despite of it, as it could not now strangle him and stop respiration. With each spoonful, however, the violence of the spasms increased; the last of the six produced extreme distress.

If I had another case, I would pass a tube into the stomach immediately after opening the trachea. Thus morphia, &c., might be given with unrestrained freedom, and the tendency to death by asthenia would not be hastened by thirst and starvation.

He now took ice pretty freely, although he was attacked with spasms whenever it was brought near him. The pulse had become slower, 84 in the minute, but much fuller than before the operation, rather bounding and distinctly unequal, but without intermission.

12.40 o'clock.—Has been lying nearly free from spasm and very tranquil, but has not slept. When attempts are made to cover his face with a sheet (natives do this before going to sleep), slight spasmodic action is produced. I now sent for some wine, but the sound of moving a spoon in the wine, which had been brought in a tin mug, produced most violent spasms—one of the worst attacks that he had yet experienced. He could scarcely be held on the bed, and uttered a distressing short moaning cry, the air passing with great force and sibilation through the canula. The attempt to give him wine was, therefore, immediately suspended,

In the absence of spasm, his pulse was not irregular, but some of the beats were perceptibly above, and others below, the average strength.

1 o'clock A.M.—*Has taken five or six spoonful of water with much difficulty.* Has had a few attacks of spasm, during which respiration was much embarrassed, although air passed freely through the artificial opening. The mind is quite clear.

2.30 A.M.—Started in a spasm when I came near his bed. *Has taken a tablespoonful of sago.* Gets spasms every few minutes. In the severest of these, excited by his dose of morphia (bringing the quantity taken up to five grains), he started back and clung to his uncle's arm with an expression of the most intense and supernatural terror, giving a vivid idea of one spectre-stricken. Pulse constantly varying, at one moment rapid and almost regular, at another slower and very irregular. No longer complains of thirst; cold perspiration on forehead and extremities. To continue the morphia every hour, providing the intellect remains clear. To have chloroform freely poured on his pillow.

3.30 A.M.—Paroxysms, not of very extreme severity, occur about every three minutes. During a moderate paroxysm, the pulse becomes more irregular than when he is at rest, sometimes faster, at others slower. To have liquid food if it can be swallowed without extreme distress. Chloroform to be freely used, so that the patient's head may be constantly surrounded with its vapour. The tube continues perfectly free.

5 A.M.—*Has taken a spoonful of milk* since last report. The frequency of the spasm is increased. Has not slept throughout the night. Pulse small and very quick, and occasionally intermittent. Had a severe spasm just now, becoming suddenly convulsed throughout the whole body. During this paroxysm, he tried to get up from the bed. His mind is not so clear as before. Continue the morphia.

8 A.M.—Has had several severe paroxysms since the last report, four or five during the last half-hour. *Took some wine and water without much difficulty.* Respiration much quicker than heretofore. Chloroform inhalation was employed, and appeared to give great relief. It rendered him unconscious of pain; and, when he recovered from the effects, he said that he felt better. The first effect of the chloroform was to increase spasm, but, when continued, it certainly gave relief: he became unconscious, and no doubt free from all suffering, and thus obtained temporary rest from the pain and nervous exhaustion caused by the constantly recurring spasm. Cold sweat on the forehead and limbs; extremities cold. Pulse small and frequent.

9.30 A.M.—Is getting spasms every five minutes. Has not taken any wine or water, but took the morphia with some difficulty. Is becoming delirious. Pulse small and very frequent. Moans and

grinds his teeth. Is very restless, trying from time to time to rise from the bed.

10.30 A.M.—Spasms more frequent. Is delirious, grinding his teeth and attempting to rise. Extremities cold and covered with sweat ; foams at the mouth and makes a terrible noise. Pulse barely perceptible at the wrist. The spasms extend to the muscles of the extremities. Has an aspect of the utmost suffering.

11 A.M.—Strong soup was injected into the upper part of the rectum by the long tube, but it was expelled by spasmodic action. The eyes have a very furious expression ; spasms more frequent. The whole body sweats ; makes a terrible noise ; is very restless ; radial pulse small and barely perceptible ; some foam in the mouth. -

12 o'clock.—Extremities cold and clammy ; pulse nearly imperceptible, spasms less violent and afflicting.

12.30.—No radial pulse, spasms weaker ; general cold sweating ; extremities cold.

1 P.M.—Died by asthenia.

No post-mortem examination could be made, as the friends insisted upon removing the body immediately after death.

This record is valuable as proving that, in a case where the horror of liquids was most intense, *opening the trachea, as was foreseen, enabled the patient to drink.*

I feel convinced that the pain of the operation added scarcely anything to the sum of the patient's agony, and was infinitely more than compensated by the power of relieving thirst, and by the very decided, though only temporary, relief of spasm and partial restoration of cardiac power which followed.

As preventing frequently-recurring attacks of exhausting trachealism, this operation must, when performed early, be regarded as tending to avert death by asthenia.

This case by no means validly tested the efficiency of the operation. The first symptoms had set in four days, or longer, when he came under treatment. There was cardiac spasm, and exhaustion was advancing when I operated.

I maintain that, *immediately a person who has been bitten by a dog exhibits distinct signs of hydrophobia, the trachea ought to be opened.*

Herbert Mayo opened the trachea of a dog, without effecting, as Youatt states, the slightest result. But was this done sufficiently early ? I have often felt strongly tempted to operate upon living animals, with a view to resolve questions on physiology and toxicology ; but have been restrained by the doubt—' would the result of my experiment, however great its interest might be, tend to the relief of human suffering ? ' I think, however, that it would be well to perform tracheotomy in healthy dogs, and then to inoculate with the virus.

This having been done, it has still to be shown whether or not the hydrophobic man or animal will remain liable to death by asthenia, the result of spasm of the heart, pharynx, muscles of respiration and general muscular system. It is questionable whether any hydrophobic patient, with the exception of Mr. Scriven's, has outlived the attack; and it has yet to be proved that death from asthenia will take place in this disease, despite judicious management and careful support, where laryngismus has never occurred.

I think that Dr. Gowers¹ speaks over-confidently in saying that 'death from *laryngeal* spasm is too rare to justify tracheotomy.' I apprehend this excellent authority to mean that spasmodic closure of the glottis, choking the patient, has not been generally observed as the immediate cause of death; but my experience teaches me that repeated fits of strangling, causing horrible anguish and inability to swallow drink and food, here appear to stand foremost among the causes of death from asthenia.

I treated cases of hydrophobia after this: but they were hopelessly advanced before they were brought to hospital.

The true nature of the disease, in Mr. Scriven's case, appears to be open to debate; still, while I may lay claim to have been the first to open the air passage, as recommended by Dalziel, Herbert Mayo, and Marshall Hall, in an absolutely indisputable case of hydrophobia, I think it probable that Mr. Scriven's was as truly a case of hydrophobia as mine was. Consequently, the priority must be claimed for him.

In the chapter on 'Tetanus'² I have alluded to Professor Verneuil's success in performing tracheotomy, when death by laryngismus has been threatened in that disease.

This operation demands care on the surgeon's own account. Considering how often slight abrasions on the hands and face pass unnoticed, the risk of being freely sprinkled with the blood and sputa of a hydrophobic patient is by no means trivial. M. Decroix, a French veterinary surgeon, proved by experiment that the saliva of a man labouring under hydrophobia is capable of exciting rabies in a dog.³

Commenting upon the above suggestions, Dr. G. N. Pitt observed⁴ that, in a case of hydrophobia, which had been under Dr. Wilks, he had the exceptional opportunity of observing the larynx during one of the spasms. He was examining the larynx in an interval between the spasms, and had the cords full in view when a spasm came on. In-

¹ Quain's Dictionary, Art. 'Hydrophobia.'

² Page 425.

³ 'Commission sur la Rage,' *Dublin Medical Press and Ranking's Retrospect*, vol. xxxix. p. 82.

⁴ *M. T.*, June 20, 1885.

stead of the vocal cords coming together as he had expected, they moved apart and remained, for some seconds, in a state of spasm in contact with the wall of the larynx. Dr. Felix Semon has pointed out that this is really what might have been expected *à priori*, if, as is most probably the case, the spasm of the larynx in hydrophobia is due to irritation of the respiratory centre; for it has, he remarked, been shown experimentally that, when this centre is stimulated, the result is powerful abduction of the vocal cords, which is explained by a close and intimate connexion between the centre presiding over the abductors and the respiratory centre. Dr. Pitt cites Niemeyer's description to the effect that the state in question is one of apnoea at the end of inspiration and not of laryngeal obstruction; he says, 'During a spasm, the thorax rises interruptedly and remains in the position of deepest inspiration for ten or twenty seconds. During this time, the features betray anxiety and terror, the eyes protrude, the head and shoulders are thrown back, then comes an expiration, with which the attack passes off.' In his case, Dr. Pitt found no evidence that the glottis was closed during the spasms. He considers that the above observations prove that there is not spasmodic closure of the glottis in hydrophobia, and that, consequently, tracheotomy need not be repeated in future. He cites the high authority of Sir T. Watson who, after watching four cases of hydrophobia, concluded that the patients died from asthenia, and not from asphyxia. My contention is that, in cases like that of Reyda Chand Dutt, the patients die from asthenia mainly caused by repeated attacks of laryngismus. Dr. Pitt concluded by expressing a hope that, after the additional evidence which he adduced, the profession will admit the wisdom of Sir T. Watson's remark that, should he become the victim of hydrophobia, no one, if he could help it, should make a hole in his wind-pipe. I replied that, in association with the observations of Niemeyer and Felix Semon, the abducted condition of the vocal cords, seen by Dr. Pitt, claims the attention of all future investigators. Still he and every one who has witnessed the universal agitation of the muscular system which attends the hydrophobic paroxysm must, I think, allow that a single observation of the state of the glottis 'for some seconds' by no means disproves the belief, entertained by Marshall Hall and others, that laryngismus is a factor in the causation of death by rabies. The 'powerful abduction of the vocal cords,' now in question, is undoubtedly a condition of glottideal spasm, in consequence of which, or, at least, in coincidence with which, respiration is suspended. Niemeyer, as cited by Dr. Pitt, says: 'During a spasm, the thorax rises interruptedly and remains in the position of deepest inspiration for ten or twenty seconds. During this time, the features betray anxiety and terror, the eyes protrude, head and shoulders are thrown back, then comes an expiration, with which the attack passes off.' Here is one of

several known conditions in which the man is said 'to die because he cannot expire.' It is evidently an arrest of respiration which, when frequently recurring, must tend to cause death by asthenia. It is, too, as Dr. Pitt demonstrates, associated with a state of laryngeal spasm which is, in all probability, not less distressing and exhausting to the system than spasmodic closure of the glottis is.

I have repeatedly seen the state described by Niemeyer; but no condition of spasm is, at once, so general and so variable as that which constitutes the hydrophobic paroxysm. In the five cases which I watched, no two paroxysms were precisely alike, save in the fact that no voluntary muscle appeared to be quite unaffected.

It has yet to be proved that, in these paroxysms, spasmodic closure of the rima does not alternate with abduction of the vocal cords. Niemeyer says, 'then comes an expiration with which the attack passes off.' So, in Mr. Scriven's case, 'the air whistled' (through the artificial opening), 'and the relief instantly afforded was obvious to all present.' If, in Niemeyer's state, the attack passes off with an expiration, we can, at any moment, obtain an expiration by opening the trachea.

Tracheotomy counteracts the ill-effects of every form of glottideal spasm, and I remain convinced that, by ensuring the hydrophobic patient against such spasm, we confer upon him a benefit second only in validity to that which would result from the abolition of cardiac spasm.

In a native adult suffering from *Chorea*, to whose case I shall make further reference at page 445; I watched the approach of choking, by laryngismus, for upwards of half an hour. Free respiration was established immediately the trachea was opened. We have seen that, in *Tetanus*, Professor Verneuil has employed tracheotomy 'with some remarkable recoveries where death from laryngismus has been threatened.' In *Hydrophobia* [vide p. 429], a medical man, doubtless guided by the character of his own sensations, suggested tracheotomy. In some cases of 'hydrophobia,' as in Fayrer's [p. 428] and Scriven's, the patient is able to drink; but, in the latter case, choking, threatening life, was excited by an attempt to drink. Here tracheotomy at once put an end to the peril of choking, and doubtless saved life. In my own case, I felt justified, as a clinical teacher, in declaring, in writing, my opinion that *tracheotomy would enable the patient to drink*. The result of my action confirmed this opinion.

Here, then, we have before us the fact that—although, in hydrophobia, no other means of treatment has been attended by valid success—tracheotomy has been followed by more or less distinct relief in repeated cases of hydrophobia and of other allied maladies.

If it be admitted, as it must be, that tracheotomy sets entirely aside the laryngismus of these diseases—placing the larynx virtually as much out of the system as if it had been removed by operation—and thus withdraws one of those agonising factors which combine in leading up

to death by asthenia, I hold myself entitled, while acknowledging the value of Dr. Pitt's fact, to maintain my advocacy of this operation.

No two men in their senses could well have been more unequally endowed with intellect and judgment than were Sir Thomas Watson and the poor Hindoo lad upon whom I operated. Still the latter had more practical experience of the effects of tracheotomy in hydrophobia than has happened to fall to Sir Thomas and to Dr. Pitt. His aspect, as he swallowed his first tablespoonful of water, was such as to convince me that he would not have agreed with Watson in declaring 'no one shall make a hole in my windpipe.' I certainly am not in accord with Dr. Pitt in the opinion that his welcome fact teaches us that 'the operation need not be repeated.'

My operation was done when the patient was greatly exhausted, and I hold that the value of Marshall Hall's recommendation will remain untested until the trachea shall have been opened in a vigorous patient who, having only just begun to show unmistakable symptoms of hydrophobia, has not yet begun to sink under those agonies which lead up to death by asthenia or laryngismus.

Convulsions.

This condition is shown, by the old returns, to have been of considerable prevalence among the children of European soldiers in Madras. We find, in Hugh Macpherson's tables, that, in the European army serving in Bengal in the four years ending 1854, there was one instance of convulsions (nature not specified) in a soldier, ending fatally; there were two cases in women, one fatal, and 181 admissions of children, with 111 deaths, or $61\frac{1}{3}$ per cent. of treated. The returns for 1880 give, for European soldiers' children, throughout India, 119 admissions and 91 deaths, being 15·7 admissions and 11·98 deaths per 1,000 of strength. In 1881, there were 102 admissions and 64 deaths, 15·6 admissions, and 9·77 deaths per mille of strength. In the following year, the numbers were—Admissions, 79; deaths, 52; proportion of admissions to strength, 12·7; of deaths to strength, 8·38. In two of the three years, the largest proportion of all deaths to strength occurred under this heading. The cases which I saw in India were mostly those in which eclampsia is an expression of pernicious malarious fever. Probably in consequence of studying manuals by English writers, European ladies in India used to give themselves much distress and their medical attendants some trouble, imagining that, as long as their children had a tooth to cut, the danger of convulsions was imminent. Still (perhaps because I watched most carefully the progress of dentition of every infant under my care), I never happened to witness the occurrence of convulsions in teething in India. A fine English girl, having been held by a native nurse for

some hours daily when a few days old, had a kind of fit which appeared to result from drugging by opium, an atrocity to which these women are sometimes addicted. She throve well, but, when in her third year, in England, being apparently in good health, she had more than one violent attack of convulsions, which yielded readily to bromide of potassium.

In native children, convulsions not infrequently appear to arise from irritation caused by intestinal worms; but the extreme prevalence of worms among the poor in Lower Bengal throws doubt upon the ætiology of many of these cases.

I have already spoken of the *Treatment* requisite in *convulsions attributable to malarial influence* (page 175). The necessity for employing anthelmintics and free gum lancing, in cases where their use is indicated, need scarcely be insisted upon. Our statistics do not show that we have yet acquired much power in treating infantile convulsions, but the following extract from a case which is given in the Madras Medico-Topographical Reports goes far to explain the fact that, out of 14 soldiers' children who were treated for 'convulsio' some fifty years ago, 13 died. 'The little patient, a boy of 7, died in little more than two hours from the onset of the attack. Cold water was sprinkled over the face, and some stimulants were at first administered; a vein in the arm and the temporal arteries were opened at 7 o'clock; but, even then, he had very little pulse, and appeared to be sinking fast, and very little blood was obtained; a blister to the nape of the neck and sinapisms to the feet were applied, without any effect.'

I attended an intemperate middle-aged European, living at a dock near Calcutta, in an attack of eclampsia which, although not fatal (at least on that occasion), was more intense than I have ever seen, even in cases of traumatic abscess of the brain.

Puerperal Eclampsia is of very frequent occurrence in India, especially in immature native primiparæ, when the head is long impacted in the pelvis, and the unhappy patient is subjected to the futile violence of ignorant dhyes. (See Diseases of the Puerperal State.)

Epilepsy.

The following data are afforded by the Returns of the Sanitary Commissioner, which, however, scarcely touch those classes in India among whom epilepsy is most prevalent.

1880.—*European troops*: no deaths from epilepsy, 19 invalidings; *soldiers' wives*: 7 admissions, no fatal cases; *children*: 5 cases, no deaths; *sepoys*: 95 admissions, 6 deaths; *prisoners*: 168 admissions, 14 deaths.

1881.—*European troops*: 2 deaths, 45 invalidings; *soldiers' wives*: 4 admissions, no deaths; *children*: 13 admissions, no deaths; *sepoys*: 51 admissions, 3 deaths; *prisoners*: 105 admissions, 10 deaths.

1882.—*European troops*: 1 fatal case, 42 invalidings; *wives*: 5 admissions, no deaths; *children*: 5 admissions, no deaths; *sepoys*: 76 were admitted, 2 of whom died; *prisoners*: 164 admissions with 13 deaths.

I have already spoken of some of the characteristics of *epilepsy* as it prevails in India; of its frequency in drunkards; of its occurrence in hot seasons and cessation in cooler weather and on return to England, and of its association with other evidences of malarial poisoning. In asylums for natives, *Epileptic Mania* frequently occurs, especially among opium eaters and gunjah (*Cannabis Indica*) smokers.

The following entry, in the annual report of a European corps, explains not only two but hundreds of cases of epilepsy, which have occurred in our regiments serving in India; '*both attacks followed continued drunkenness.*'

I saw many of these cases in India. Not having observed eclampsia as occurring in drunkards, during twelve years' experience at Guy's Hospital, I was, at first, inclined to form the worst prognosis in Indian cases. Doubtless, this is a very unfavourable symptom, and I have seen death occur in convulsion. Still such cases may do well. I kept in view a well-to-do Englishman who survived the first severe attack of eclampsia for fifteen years, drinking steadily, and died from another cause. I had reason to think that he was supplied with none but the best liquor.

Dr. Geddes¹ noticed that, in the fort of Masulipatam, which is encompassed by a salt marsh, 59 cases of cephalic disease, producing epilepsy, occurred in a European regiment, in four years.

I have already cited, in my remarks on the Eclamptic Pernicious Complication of Malarious Fever,² Dr. Arthur Payne's monograph on 'Epilepsy, as a result of Malarious Infection.'³ When published in 1861, Dr. Payne's observations upon the conditions of the blood in paludal cachexia were well to the front of the pathology of the day. They still deserve the attention of investigators, and it is much to be regretted that they have not, as far as I am aware, been made the basis of systematic observation in several of the malarious districts of India, and of other countries. I apprehend that the eclamptic pernicious complication of malarious fever does not present itself frequently in any locality. Thus it was lately reported⁴ that 'only one case of actual malarious pernicious fever was admitted to the Santa Spirito Hospital, Rome, during the year 1884; and only two during 1883. Consequently, a published notice of each case, as it occurs, would be as easy as it is desirable.' Doubtless, many experienced medical men would assure us that they had never met with a case. Not long ago, I heard of what I consider to have been a typical example of eclamptic pernicious fever which was absolutely misunderstood by every one who saw it.

¹ *Clinical Illustrations of the Diseases of India.* ² Vol. ii. for 1882, p. 523.

³ *I. A.*, No. 14, p. 597.

⁴ *M. T.*, vol. i. or 1885, p. 626.

We may be allowed to feel doubtful, at first, with regard to the cause of an unexpected attack of eclampsia in an apparently healthy person ; but where, in one who is not teething, there is no history of epilepsy or alcoholic excess, and no evidence of the presence of albuminuria or of intestinal worms, and where the locality is malarious, steady reading of the temperature during the attack and for several hours after its cessation, and the use of the quinine test, when the bowels have been thoroughly cleared by a calomel purge, ought to throw full light upon the majority of such cases.

In my work on Indian Medical Jurisprudence, p. 624, I have stated and discussed the fact that natives are frequently drowned by falling into tanks in the epileptic fit. Anything glittering, especially fire or water, appears to act as an excitant of the epileptic as of the hydrophobic paroxysm. ' Oft-times it ' (the dumb spirit) ' hath cast him into the fire, and into the waters, to destroy him ' (Mark ix. 22). We are told ¹ that a son of Sir James Dalrymple, first Lord Stair, was frightfully disfigured by falling on the fire in a fit of epilepsy. The following Indian notices appeared subsequently to the publication of my work :

March 28, 1871 (not excessively hot weather), a Mussulman of Calcutta, 30 years of age, who, it was said, had been for some time subject to epileptic fits, fell, it was believed in a fit, while washing in a tank at about half-past seven in the morning, and was drowned.

On April 9, (hot season) of the same year, a very aged man subject to fits, while sitting at the edge of a tank at Puddahpooker (Calcutta), was seized with a fit, fell in and was drowned.

Early in January (cold season) 1874, it was reported that ' at Kasundi, a man, after eating, went to wash his plates in Kinoo Sircar's Tank, when he fell in. It is said he had an attack of epilepsy at the time.'

The often-noticed association of epilepsy and *lumbrici* is only too common in India, where, however, as I have before noticed, the occurrence of these parasites is so frequent as often to make us hesitate to assign it as the cause of other diseases. The long-recognised liability to epilepsy of the subjects of *tape worm* ² is a subject which deserves further investigation.

It is noticed, in Dr. Gordon's edition of the China Reports, p. 202, that, at Shanghai, during the six months—October 1875, March 1876—' one man had an epileptic seizure which suggested the presence of tape-worm. A smart turpentine purge brought away an unusually perfect specimen of the parasite, and no attack has since occurred.'

¹ Nimmo's cheap edition of the *Bride of Lammermoor*, notes biographical and critical, p. 3.

² ' It ' [epilepsy] ' most frequently depends upon worms. The tape and lumbricoid worms are those which oftenest induce it.'—Copland's Dictionary, Art. ' Epilepsy,' p. 794.

Sixteen years ago, the following suggestion was made, in India, by Dr. J. Fleming¹: 'Upwards of a hundred cases are recorded, both in our own country and on the Continent, where the scolex condition of the tape-worm has infested the human body and caused death; and there is reason to believe that many cases, returned as "died from natural causes," may have been owing to the development of immature tania in the brain or in some other vital organ. Certain epileptic seizures, which, in many cases, terminate fatally, can be distinctly traced to tape-worms, and many instances are on record where the expulsion of the worm from the intestines was followed by a complete cure of epileptic convulsions. The development of immature taniæ in the brain, even one, is sure to cause death.' I believe that, had we been able to recognise this condition in my student-days, we should have found that it existed in a very powerful-looking young man who died in an epileptic paroxysm.

Treatment.—It is almost needless to insist that the free use of quinine is indicated in all cases where epilepsy appears to depend upon malarious influence. Whenever I had a case of epilepsy in my ward, the nurse had constantly at hand a large smooth mass of ice. The immediate application of this to the nucha almost invariably lessened the severity and duration of the paroxysm. Where ice is not available, the constituents of a freezing mixture, to be applied in a bladder, ought to be constantly in readiness. This treatment will doubtless be employed, prophylactically, should further observation confirm, as a natural law, the fact reported in the 'I. M. G.' for February 1866, that, in a female patient, a rise in temperature usually indicated the occurrence of a fit. The European who becomes epileptic in India (I do not speak of the confirmed drunkard who is scarcely worth sending home) should always proceed to Europe, by the long sea-voyage, without venturing to encounter another hot season. In 1830, Mr. J. R. Preston entertained the idea that ligature of the common carotid artery might be advantageously had recourse to in various diseases of the brain, especially those considered to depend upon 'congestion, inflammation, or irritation' of that organ. Among the diseases in which he anticipated benefit from this heroic measure were 'apoplexy, phrenitis, hydrocephalus, many cases of injuries to the head, palsy, epilepsy, and insanity.' He added an expression of hope that 'tetanus might be cured by tying both carotid arteries.' Mr. Preston operated in a case of hemiplegia and in one of epilepsy. As this idea has been re-adopted of late, the details of his cases, which will be found at pp. 345, 359, and 444 of the fifth volume of the 'Transactions of the Medical and Physical Society of Calcutta,' may interest the curious.

¹ 'Remarks on Cyst-Infected Meat at Meean Meer, its Nature and Prevention,' *I. M. G.*, vol. iv. p. 114.

In 1880, there was not any case of *Chorea* among the European soldiers' children in India. In the following year, there was only one. In 1882, there were three admissions. In the three years, I find the following: two European soldiers invalided for chorea; one European woman admitted; eight sepoy treated, with one death; one prisoner admitted.

A medical officer, who was an eminent naturalist, was the subject of moderate, but very marked, persistent *Athetosis* (I then called it 'Chorea'). He carried it off very lightly, and I never presumed to question him upon its history. Much of his time was spent in the field; and, otherwise, he appeared to be remarkably athletic and healthy, but he did not attain old age.

As I have mentioned, I never saw *chorea* in an English child in India. Acute rheumatism is rare in Lower Bengal. I had one choreal patient, a native girl. In the ward next to mine, Dr. Chuckerbutty had a native man of middle age, in whom universal choreal movements caused extraordinary rapid contortions of the features and limbs, giving him a most quaintly hideous appearance. He was suddenly attacked with spasm of the glottis. Tracheotomy was performed; he died from ichoræmia. As this was not my own case, I was unwilling to operate, but the tendency to death by laryngismus was so strong that the half-hour during which I watched his sufferings, with the instruments at hand, was one of the unpleasantest in my life. The introduction of the tube at once relieved all distress in breathing.

Hysteria will be considered in the section on Diseases of Women. *Catalepsy* (hysterical) was markedly displayed in a case which I attended in England; I never saw or heard of the occurrence of this condition in India. For *Neuralgia*, vide the chapter on *Diseased Conditions consequent upon Malarial Cachexia*, p. 259.

I have seen *Spinal Apoplexy* twice—in a specimen brought by Dr. Clendinning to an early meeting of the Pathological Society, and in a native man who was picked up moribund on the road. In this case, there were two large recent clots in the substance of the cord. A traumatic case, which probably originated in this manner, is represented by Prep. 801 in the Calcutta Medical College Museum. 'Laceration and softening of the spinal cord near the corda equina, caused by a fall on the back from a height of ten or twelve feet. The woman died paraplegic, with a fistulous opening leading from the bladder through the abdominal parietes, just below the navel, from a patient of Sir Joseph Fayrer.'

Spina Bifida has been occasionally seen in natives. Recorded cases would probably be more numerous if these lives were not so extremely precarious.

Dr. C. Finch¹ treated the child of a Brahmin, aged three months,

¹ *I. M. J.*, N.S., vol. v. p. 350.

for lumbar spina bifida. The tumour was as large as an orange, the infant was emaciated and small. It was frequently noticed that, on the removal of fluid by puncture, the anterior fontanel became depressed. After an interval of two months, fluid began to exude through small ulcerations. There was increasing hydrocephalus. The removal of half an ounce of fluid, by puncture, was followed by symptoms of fever under which the child sank. In a European child, attended by Dr. Finch, the covering of the protrusion, which was dorsal, sloughed and separated when the child was a month old. It became convulsed, and died almost immediately.

Preparation 1209 in the Calcutta Medical Museum Catalogue shows spina bifida, presumably from a native subject.

Assistant Apothecary G. W. Eate has reported the case of a native child, a month old, in whom he treated a lumbar spina bifida by puncture and iodine injection with success. Assistant Apothecary Mitchell¹ has described the case of a man, aged 21,² caste Rajpoot, 5 feet 2 inches in height, pale and emaciated, who was born with a tumour, as large as a pigeon's egg, projecting from the upper part of the sacral region. He stated that it gradually increased from infancy, and that it suddenly broke, discharging an enormous quantity of pale fluid. When this occurred, he was obliged to keep to his charpoy (bed), but, subsequently, he had walked well. In ten days, the tumour had resumed its original size; pain was not felt in two days during which the sac was discharging, but he used to be sick at stomach, and had giddiness while it was refilling. [How interesting it would have been to observe precisely the condition of an adult who had suddenly lost a large proportion of the cerebro-spinal fluid!] A tumour, of the size of two closed fists, was found depending from the lumbar and sacral regions. It was very tender and painful to touch, and its integument was 'of dark hue.' He had a strange feeling of cold running along the spine to the head, which he attributed to excessive weakness. He complained of giddiness, and answered at times incoherently. The pulse was soft and regular. He had walked from his village (twenty-two miles) to seek aid. This had caused pain and exhaustion, and a feeling of great weight over the tumour. Mr. Mitchell immediately punctured the sac, drawing off some fluid to relieve tension. On the following day, another puncture was made and brought away twenty-seven ounces of fluid. A slight compress and bandage were applied. There were no bad symptoms with the exception of slight giddiness and a little heat of skin and excitement of pulse with excessive thirst.

¹ *I. M. G.*, vol. ii. p. 122.

² Mr. Holmes says that, in a published case, the patient survived to the age of fifty, and he has heard of older cases in private. See Holmes's *Surgical Diseases of Childhood*.

He did very well for two days, looked cheerful, and anticipated speedy recovery, but the sac filled again and became larger than it had ever been before, and the patient began suddenly to sink. [Here the account is not clear, but there appears to have been trismus.] ‘There was a difficulty in swallowing, and spasms of the face and throat, caused no doubt by the violent curvation backwards of the trachea and œsophagus.’ The tongue, lips, and teeth were covered with sordes, and an unhealthy redness (no doubt erysipelatous) was spread over the tumour. He expired six days after the first puncture. The friends would not allow an examination *post-mortem*.

I saw something of *General Paralysis* in India, both in natives and Europeans. One case, that of a member of the Civil Service, concerned me much, as I liked him, and saw and heard a good deal of him for seventeen years. When I knew him first, he was a very highly educated young man, and was regarded by the senior civilians as singularly well up in his work. His manners were those of a clever and very quiet person. Still, everyone who knew him told stories of his eccentricity and oddity. He was absent-minded, and, on two occasions, when handling fire-arms in a slovenly manner, wounded servants. In about the sixth year of his residence in India, he was sent home insane. He recovered, and continued to serve, but was not entrusted with very important charges. In about the nineteenth year of his service, I found him still at work, but much altered in appearance and manner. His face bore very markedly the characteristic stolid flabbiness of commencing general paralysis, and his laugh, which had been pleasant, was abrupt and fatuous. Still, during an evening which I spent with him, I could discover no evidence of mental failure. Shortly after this, I received an official report from him to which he had affixed his signature upside down. I returned the document with an entreaty that he would be careful. For some years, he was prone to be cruel, as if in joke. Finding the children of the European residents standing at a fence listening to a military band, he burnt their noses with his cheeroot. His irrational persistence in torturing the pet dog of a high official led to inquiry and to his suspension. He became generally paralytic and imbecile. I am confident that he was not a drunkard when I first knew him, but I believe that he had recourse to alcohol when he felt depressed.

It has been remarked by Dr. Wise, jun.,¹ that one of the most important points connected with insanity in India is the rarity of general paralysis. In the Patna Asylum Report for 1866, the deaths of two Patna women, aged respectively 26 and 35 years, are recorded. With this exception, no other cases are cited in the reports of the Dhullunda, Patna, and Moydapore Asylums between 1862 and 1867. In the Dacca Asylum, Dr. Wise was only able to discover three cases among

¹ *I. M. G.*, vol. iv. p. 75.

1,576 admissions and 925 deaths. This infrequency, he insisted, becomes more unaccountable when the records of European Asylums are contrasted with those of Bengal. He cites the statement of Calmeil that there is one general paralytic in every 15 male (lunatic) patients, and 1 in 50 among women. Foville calculated 31 general paralytics to 334 insane, or 9·2 per cent. According to Bayle (1855), the proportion of paralytics in the asylums of Paris was 1 in 4; but Baillarger gave, for Bicêtre and Salpêtrière together, the proportion of 1 in 16. Dr. Wise thought that, if search were made in the case-books of the different asylums, cases would be found, classed under the head of chronic meningitis, which were really cases of general paralysis. He gives the histories of the only cases (natives), 4 in number, which had occurred in the large asylum at Dacca during the preceding 27 years.

In 1880, 1 European soldier died of general paralysis, and 2 were invalided; 1 fatal case occurred in the native army, and 2 prisoners were under treatment.

In 1881, no case, in a European, was recorded; 2 sepoys were admitted, and one prisoner died.

In 1882, the disease does not appear in the European returns; a sepoy was treated, and 1 case (fatal) occurred among the jail population.

The ordinary diseases of the *Spinal Cord and its Membranes* are seen in natives and Europeans. Three interesting forms of *Spinal Paralysis*—Paraplegia, Locomotor Ataxy, and Lathyrism—occur.

Paraplegia.—At and near Chittagong, on the east sea-board of Bengal, a form of equine paraplegia, known as *kumree*, is so prevalent that, as a general rule, horses are not kept there. Burmah ponies which, if let loose, might find their way home across country, do well in that climate. I saw the disease, at its very commencement, in a valuable horse, and, towards its close, in a favourite animal, which lived long and grazed comfortably in a field, dragging the hinder half of its body after it with an appearance of extreme paralysis.

I believe that this disease does not originate in Calcutta or in the Upper Provinces, but it occurs in various Indian localities within the influence of the sea-breeze. Thus, in the report of Travancore¹ we read: 'Horses here, as well as all along the western coast, are very liable to become weak in the loins, particularly if exposed to the wind when much heated. Castration is often resorted to as a preventive, and it seems to be generally admitted that geldings suffer less frequently than entire horses; when attacked with this complaint, the animal is rendered completely useless.'

In the report on Quilon, a marshy district,² we are told: 'Horses in this climate are very liable to become weak in the loins; the nature

¹ *The Medical Topography and Statistics of Madras*, 'Travancore,' p. 89,

² *Ibid.* p. 113.

of this disease is not correctly understood, but it appears to be an affection of the spinal marrow causing paralysis, or an approach to it, of the animal's hind quarters. A permanent cure has seldom if ever been effected. Castration, as mentioned in the report for Trevandrum, would appear to be the best preventive, mares and geldings being very rarely attacked by the disease. This disease is most frequent during the land winds in November, December, and January.'

Those who may be desirous of going further into the literature of this malady will find many interesting particulars in papers by Messrs. Breton and Twining, at p. 337 of the first volume of the 'Calcutta Medical and Physical Transactions,' and in Dr. Shortt's remarks on the *kumree* of Orissa, No. ix., p. 187, of the 'Indian Annals of Medical Science.'

Bontius and his followers described *Barbiers*, from which that sage old Dutch physician himself suffered. He attributed it to 'a thick, viscid, pituitous humour *seizing the nerves at night*.' An article, under this heading, will be found in Copland's 'Dictionary of Medicine.' All paralyzes of the lower extremities appear to have been thus designated. The word is no longer used, at least in India. A '*Stroke of the Wind*' is frequently spoken of. A well-known steamer captain, who had been in the habit of sleeping all night in the open air for years, was, at length, stricken in this manner on shore. I experienced the intense misery of a moderate attack. One of my windows having been blown open in the night, at a time when I was in low health, I found myself lying on my side in a chill damp breeze. Every voluntary muscle in my body seemed to be painfully cramped, and I was unable to move a single joint. After a minute or two of great suffering, I found that I had recovered the use of my right hand and forearm. By degrees, I chafed the other arm into action, and was then able to rub my body and lower limbs, and so regained command of their movements. In the morning, only some stiffness remained; but, from that time, I have been subject to muscular rheumatism. Had I been exposed during the whole night, the result would, I believe, have been *barbiers* or paraplegia. Henry Marshall saw many cases of *barbiers* among the Caffres of the 4th Ceylon Regiment, in 1812. He did not notice it among the Cingalese. He met with it in Europeans in Ceylon, and observed an analogous affection in horses and dogs; from which they were never known to recover. The cause was evidently chill, especially in sleep. At the onset, there is generally partial paraplegia with pins and needles in the affected limbs. In the severest cases, the rest of the body becomes paralysed and deprived of both motion and sensation. The limbs become contracted, nutrition fails, and the patient sinks.¹

I have noticed the loss of power in the lower limbs, which is a

¹ Dr. W. J. Moore's observations on 'Maladies Attributed to Lunar Influence, Rheumatism, Paralysis, Ocular, &c.,' *I. M. G.*, vol. iv. p. 180.

complication of beriberi, at page 71. Much of the paraplegia seen in Bengal is attributed to chill; cases also result from meningeal apoplexy, morbid growths, sclerosis, syphilitic disease, and other ordinary causes. In 1880, one European soldier was invalided for paraplegia, 26 sepoy were treated, with 6 deaths, and there were 18 admissions with 4 deaths amongst the prisoners. In the following year, 1 European soldier was invalided; 23 native soldiers were admitted, of whom 6 died; 10 prisoners were admitted, with 5 deaths.

In 1882, 2 Europeans were invalided. There were 18 admissions with 4 deaths among the sepoy; 7 prisoners were admitted, of whom 3 died.

I saw four cases of officers who recovered from paraplegia. Two of these gentlemen have remained healthy for many years, but one of them has lately died, after some return of paraplegia, at the age of seventy. The other two were free livers. I met one of them, walking actively, at Charing Cross, but he did not live long. The other was fatally attacked with dysentery a few months after his return to Europe.

I had a very well-marked case of *Locomotor Ataxy*, in a native man, and saw a few others. In 1880, the number of European soldiers invalided for locomotor ataxy was 4. The admissions of sepoy were 10; of prisoners 8, one fatal. In the following year, 5 European soldiers were invalided for this cause. Sepoy, 3 admissions, 1 death: prisoners, 2 admissions. In 1882, 6 Europeans were invalided; 11 native soldiers were admitted; no prisoners.¹

Lathyrism.—Palsy of the lower limbs, caused by eating the *dal* of a lentil, *lathyrus sativus*, prevails extensively in Upper and Central India, especially near Allahabad and in Upper Scinde.²

Recently, M. Proust has observed this disease in the Jurjura mountains of Algeria.³

I saw, but had nothing more to do with, two cases of *Pseudo-Hypertrophic Paralysis*, in a native male child in Calcutta and in a young native man in Bombay.

Progressive Muscular Atrophy.—A native lad was brought to me in whom this malady was advancing so very characteristically that I showed it to my class as a typical case. A well-marked case, occurring in a native surgeon, is given in the 'I. M. G.' for June 1874, by Dr. K. M. Downie.

In his valuable commentary on *Functional Paralysis in Children*,

¹ See a case of *Locomotor Ataxy* in a sepoy, with valuable remarks, by Mr. B. Evans, *I. M. G.*, vol. iv. p. 189.

² The descriptions of *Lathyrism* given by Buchanan-Hamilton, Sleeman, Kinloch Kirk and James Irving are compiled in my *Medical Jurisprudence* for India. Dr. Irving's leading papers will be found in the *I. A.*, No. 12, p. 424, and 23, p. 89.

³ *Lancet*, July 21, 1883,

already cited at p. 389, Dr. A. Garden observes: 'If this change [wasting] in the muscular structure is a diagnostic point of myogenic paralysis, but few of the cases' [of infants here] 'commented on can be classed as such, nor can this form of paralysis be considered as one of childhood alone, for there is a class of cases, by no means unfrequently met with in adults, in which all the diagnostic points, such as limited locality and wasting of the muscles, without apparent change in the nervous system, exist; for instance, the common paralysis of the deltoid and supra- and infra-spinatus following injury, excessive use or rheumatism, and leading rapidly to atrophy and wasting of these muscles, of which I have seen nine or ten cases amongst natives within the last few years.'

Neuroma.—Under the head of malarial cachexia (p. 261), I have spoken of certain forms of this disease. Knots on the nerves leading to the anæsthetic parts are almost invariably characteristic of leprosy.

Sir Joseph Fayrer has given¹ a description of the structure of a neuroma of the ulnar nerve, fusiform, and about as large as a pigeon's egg, which he removed from a middle-aged, healthy-looking Hindoo. It was firm and very painful to the touch, causing numbness and shock up the arm and in the hand on the least pressure. It had been of very slow growth. It appeared to consist of dense white fibrous tissue in which the nerve fibrils were distributed, not equally throughout, but so much as to render separation of the nerve trunk from the tumour impossible.

Mr. Browning² treated a Hindoo male, *ætat.* 35, in whom a tumour, of the size of a small orange, could be felt under the tendo Achillis. It was of ten years' standing, and was so very painful that he was unable to work. It was found that there was a second tumour as large as a pigeon's egg. These were neuromata intimately connected with the posterior tibial nerve and its division into plantar branches. The neuromata lay in separate sacs having a glistening lining, like a serous membrane. The masses were, centrally, soft and shreddy, and were finer towards the surface. The epineurium stretched over the tumours, formed a tough fibrous sheath; no nerve-fibres, with the exception of a few superficially, could be detected.

Facial paralysis is always likely to attack those Europeans who are so imprudent as to sleep in open verandahs during hot, damp, and changeable weather. I only recollect to have seen two cases in Bengalis, probably because they sleep with their faces covered. In one of these, the poor fellow had been greatly exposed in a storm.

I am acquainted with one case of *Tic convulsif* in a lady resident

¹ *I. A.*, No. 16, p. 310.

² 'Report of South Indian Branch of the British Medical Association,' *I. M. G.* November 1885.

for many years in India. It was occasioned by mental shock, and has, I believe, been permanent.

I saw a well-marked case of *Scrivener's palsy* in a surgeon, and another in an English portrait painter and drawing master, long resident in India, who was diabetic. The returns for 1882 give the case of a sepoy who was treated for this disease.

Cases of *Paralysis* from (1) *Lead* and (2) *Arsenical Poisoning* in India will be found at pages 293 and 126 of my 'Medical Jurisprudence for India.'

Sciatica.—I do not find any allusion to this affection in the Sanitary Commissioner's returns for the three years ending 1882. In Lower Bengal, I saw a very few cases. In one or two, the attack was ascribed to sitting on a damp seat. I have alluded, in the chapter on Tetanus (p. 421), to the case of one of my patients, an East Indian woman, in whom an attack of tetanus was preceded and attended by idiopathic sciatica.

In a case of neuritis of the sciatic, in a native gentleman, setting in almost immediately after salivation for a venereal affection, Sir Joseph Fayrer, detecting deep-seated fulness and fluctuation at one point in the course of the sciatic, made a puncture with a long narrow knife down to the indurated part, and gave exit to more than half-an-ounce of clear serum. There was immediate and almost perfect relief; recovery was speedy.¹

Dr. James Irving has recorded² the very obstinate case of a strong healthy gentleman in whom, after most other means had failed, cure was effected by applications of ice all over the affected hip and thigh.

Dr. Debove³ has obtained a rapid cure, in several cases of obstinate sciatica, by applying chloride of methyl, in the form of pulverisations, along the course of the nerve. The chloride reduces the temperature to 23° below zero, Cent.

Mania.

I have so fully discussed the various forms of *Insanity* as it occurs in India, in my work on 'Medical Jurisprudence' (pp. 774–829), that I need only give a few additional notes in this place.

I have already mentioned that, in English regiments, when the men drink freely, there is a great deal of *Acute Mania*, which, like epilepsy, occurring under the same circumstances, is generally recovered from when the patients are sent to a cool climate and are kept sober.

Dangerous injuries to the skull and attacks of heat-stroke are frequent causes of mania in India. Important facts bearing upon this point will be found in the chapter on *Heat-stroke*.

¹ *Observations*, p. 590.

² *I. M. G.*, vol. iii, p. 277.

³ *Gazette des Hôpitaux* and *M. T.*, vol. i, for 1885, p. 292.

I think that it must be added that drink-craving is not rare as a result of traumatic injury to the head and of heat-stroke.

A large proportion of the inmates of asylums for natives are found to be habitual gunjah-smokers and opium-eaters, and many of these are epilep^h'ic. Insanity, from the use of intoxicating drugs, is to be expected in a country where the Pindaree soldiers and their horses were drugged, whenever desperate attacks were to be made; where the women who underwent *sati* on their husbands' pyres were previously drugged, and where the assassin and he who resolves upon running amock are always primed with *bhang* or opium. In his report on the Bengal asylums for 1869, the Inspector-General of Hospitals observed that 'among the recognised causes of insanity, the table shows that intoxicating drugs or liquors formed, as usual, the principal excitants of disease. In 1868, a percentage of 37·4 of the total treated was recorded under this head. During the present year, 35·2 is the proportion, gunjah, and bhang alone forming 29·1 per cent. of the total in the preceding [year], 30·7 per cent. in 1869.'

It appears¹ that, of 166 cases in which the predisposing cause of insanity was ascertained in patients admitted to the asylums of Bengal Proper, 107 were attributed to the excessive use of gunjah or bhang, 13 to undue indulgence in drink, and 2 to opium eating. Of 44 cases admitted or re-admitted to the Dullunda Asylum, 28 were attributed to the use of preparations of gunjah. In Patna, 41 out of 53; in Dacca, 16 out of 32; in Cuttack, 7 out of 8; in Berhampore, 7 out of 10; and, in Hazareebaugh, 8 out of 9 belonged to this class. Of 410 lunatics from this cause treated in the asylums, 56 completely and 51 partially recovered. While it is most unsafe to venture to deprive the habitual opium eater suddenly of the whole of his noxious supply, my maximum daily allowance of the drug was never allowed to exceed nine grains of pure opium. I never observed any ill consequence from my invariable practice of immediately withdrawing the supply of gunjah.

In his report for 1869 of the Dullunda European Asylum, Calcutta, Dr. Payne remarks: 'It is observable that intemperance has been the imputed cause in an unusually small number of cases this year, in seven only of the total number (78). I have, in former years, recommended that statements with reference to this cause should be received with great caution, as intemperance, as an early manifestation of insanity, is so common that it is often mistaken for its precursor' [cause?], 'and it will, I think, be generally found that the more carefully inquiry is made into the antecedents of insane persons, the less will be the proportion of cases whose origin is ascribed to intemperance.'

¹ Government Resolution on the Report on the Lunatic Asylums in Bengal for the year 1877.' Supplement to the *Calcutta Gazette* for August 14, 1878.

There is another fact, of which I have had ample experience, which is not sufficiently recognised either in society or in military courts of inquiry. It has been frequently observed, especially of artists and literary men who have lived by sustained mental toil, 'Great as his talent was, he degenerated into a confirmed sot, and died mad or apoplectic.' Here the true sequence and meaning of events is overlooked, sadly to the detriment of the unhappy man's moral reputation. The fact is that—the unnaturally overworked brain having been found, for a time, capable of rallying under the influence of alcoholic stimulants—these were habitually resorted to until the worn-out nervous system failed under the double strain of labour and intoxication. It is clear that, in inquiring into a plea founded upon this law, sensible men will not allow that it can be taken as an excuse for the mere sensual excess of the idle low-minded sot.

In my work on 'Medical Jurisprudence' (p. 829) I alluded to the practice among Indian criminals of making cat's-paws or tools of 'softies'—persons of decidedly and notoriously weak intellect, but gifted with a degree of unscrupulousness and cunning which, as qualifying for the commission of criminal acts, almost compensates for the higher faculties in which they are deficient. In this day of conspiracies, the following note, from Granger, may deserve a glance. Richard Langhorne, who was executed for treason in 1679, engaged, a little before the Restoration, a half-witted person to manage elections for him in Kent, and was asked by one who was privy to the secret why he employed so weak a man in that business. He very frankly replied that it was a maxim with him to employ men of this character, because, if such agents should take it into their heads to turn informers, it would be easy to invalidate their evidence by representing them as madmen.

Of late, there appears to be a disposition in the English mind, if not in our courts of justice, to demand that the homicidal maniac shall only be spared on proof that he 'did not know what he was about' when he perpetrated the act. This might be, but is not received as a plea for the homicidal inebriate; but its application to the subjects of several kinds of madness betrays absolute ignorance of the first laws of insanity. The subject of acute mania may strike in unreasoning frenzy, as Elia's Mary Lamb did. Here there is no criminal responsibility, and restraint is the only just award. But crime is committed, under delusion, by undoubted maniacs just as calmly and thoughtfully, perhaps with a greater amount of steady deliberation, than murder for revenge or plunder is by the ordinary criminal. Thus, the first evidence of insanity in an accomplished and amiable medical officer in India, was that, at his usual morning visit, he prescribed, for every one of his numerous sick, a large dose of Glauber salts. When asked his reason for this novel practice, he

calmly explained that he gave sulphate of soda as an antidote to carbonate of lead with which the Devil had poisoned his men. When this courteous, gentle-mannered officer requested me, as Secretary to the Medical Board, to inform him why he had been dismissed from his pleasant appointment to be sent to England when in perfect health, I found it difficult to answer him. I feel very little doubt that, had it appeared clear to him that it was to his child's good to remove it from the trials of life, he would, composedly and with equal deliberation and pain, have acted upon that conclusion. Here, again, had this unfortunate practitioner imagined that belladonna had been the poison employed and thus had killed the whole party with antidotal doses of opium, a sentence of restraint would have met all the exigencies of the case. There is, however, a class of homicidal maniacs who are, as I believe, so unquestionably subject to the fear of the law that, in the interests of humanity, they ought not to be exempted from its punishments.

Not long before I left India, I had, placed in my hospital for observation and report, a middle-aged European who was in custody for firing at a public character in a coffee-room. Although he was undoubtedly the subject of maniacal delusions, he was very soft-spoken, and reasoned upon certain false premises with the calmest deliberation. He told me that the act of violence was quite accidental; there was ample evidence that it was not an accident. I asked him why he carried a pistol—he replied that, lately, he had been in so much risk of being run over in Tank Square that he had determined to shoot the horse should the like occur again. I replied that, under these circumstances, the horse would not be to blame, and that he would, at least, have to pay for killing it. He then said that, being an usher in a school, he considered that the sight of a pistol on his desk would enhance his authority over the boys! While with me, he remained quite free from all appearance of excitement. He died of dysentery soon afterwards in the jail.

A man of education, one of a party of recruits of whom I had medical charge on board the transport in which I first went to India, then appeared to be a quiet and well-behaved person, generally regarded by his comrades as a 'softy.' Twenty years afterwards, he was sent to me for observation. He had been taken into custody for having endeavoured to break into a relative's house armed with a deadly weapon, and then having completely wrecked his own dining-room. On being questioned, he said, with an air of the greatest mildness and good temper, 'I am sure that you will not blame me when you hear the facts!' Returning home from office tired and hungry, I found the table laid, but my wife was out. I certainly felt vexed, and, believing that she was at her sister's, I ran out. Recollecting that the door was generally padlocked, I caught up my gun

barrels, with which I endeavoured to break the fastening. Failing, I went back and, feeling provoked, I drew the corner of my table-cloth off and broke the crockery. I then broke the furniture with the barrels. They say that I was mad ; I deny it, I was only provoked, and, I think you will allow, with reason.' The professional man, with whom he had worked for years, said that he had an excellent knowledge of his business and received a large salary. He remained for a considerable time under my observation, being always quiet in manner and civil-spoken ; but he, at length, displayed such unmistakable and troublesome signs of mania (satyriasis) that we were compelled to remove him.

Wolves kill in obedience to the dictates of their instinct and appetite, but are powerfully deterred by a sense of personal danger. As long as the shooting of these animals is considered advisable, I would hold such madmen as the wielder of the pistol and of the gun-barrels subject to the terror and penalties of the law—hanging them without remorse when they dare to commit murder.

In such homicidal maniacs, the sense of self-preservation is fully alive, and their consciousness of the peril of wrong-doing is as strong as that of the cat or the pickpocket.

Osteo-Malacia of the Insane.—Cases of rib-breaking in lunatic asylums have been reported in all the three Presidencies of India. I have cited the case of a native at page 442 of my 'Medical Jurisprudence.'

DISEASES OF THE EYE.

My remarks will be almost exclusively confined to the chief of those peculiarities which distinguish ophthalmic disease in India.

It appears, in the Report of the Sanitary Commissioner for 1881, that the admissions for eye diseases during that year were, among the European troops throughout India 14·1, and among the native soldiers 25·9 per mille. Among the jail population, the ratio was 13·7. The highest rate was among the women and children of European troops, of whom 30·2 per mille were treated for ophthalmic disease.

Orbit.—In December 1880, Baboo Devendra Nath Dey brought before the Calcutta Medical Society a case of *Epithelioma* of the orbit disorganising the eye. On excising the eye, the growth was found to be attached by a broad and rugged base to the orbital plate of the frontal bone.

Eyelids.—The *Blepharitis* of the present day, which I used to know as *Ophthalmia tarsi*, being especially a disease of fair-complexioned and strumous people in cold climates, is not common among the dark races of the plains of India. I watched, for many years, the cases of two aged gentlemen suffering from *Entropion*. They deserve notice

for the benefit of those who may refuse to submit to cure by excision of skin. One of these individuals always wore a strip of black sticking-plaster which slightly everted the lid. The other, a medical man, who had suffered greatly from facial neuralgia and from ophthalmia tarsi, experienced years of mental torture from what he conceived to be malignant disease of the antrum, the inverted lashes occasioning great conjunctival irritation extending downwards through the nasal duct. All trouble ceased when the plan of keeping the lower lid free from ingrowing hairs, by the use of tweezers, was adopted.

Mr. Macnamara removed an *Epithelioma* from the skin of the inner angle of the left eye of a clergyman in India, whose father was considered to have died of cancer of the face. No reproduction of the disease had occurred when the patient died of fever five years subsequently.

Warts, growing from the skin of the lid and from its tarsal margin, are frequent in India. I have usually twisted them off, but they generally disappear spontaneously when left alone.

Mr. Albert Leahy¹ removed a *Dermoid cyst* from the upper eyelid of a Brahmin, aged 33. It had grown with his growth. It lay mainly beneath the orbicularis palpebrarum. It projected backwards for about half an inch beneath the supraorbital ridge, and was attached to the periosteum at the margin of the orbit. It contained a quantity of dark green fluid loaded with albumen, and of the consistency of castor oil. The cyst wall was about a quarter of an inch thick, and was composed of layers of white fibrous tissue. Lying loose at the bottom of its cavity, was a coil of hair about the size of a shilling. The coil was regular and the hairs composing it were about $2\frac{1}{2}$ inches long.

I have noticed, at p. 357, the case, by Dr. Chatham Gray, of a native boy of 12 suffering from *Molluscum*. The eyelids were especially affected. One eye was lost, the other was almost disorganised.

Functional Ptosis occasionally results in India from imprudently sleeping in the open air. In the following case, ptosis was clearly an early symptom of rapidly destructive brain mischief. At the time of the Mutiny, I saw too much of an intemperate officer in the prime of life, who gradually fell into a state of mental degradation which appears not to have been considered such as to justify his removal to the lunatic asylum. One very hot day, I met him in a shop looking unusually clean and sharp; at a second glance, I saw that he had ptosis of one eyelid. In a day or two, it was reported that he had died suddenly.

I treated for some time, but not to its termination, the case of a native suffering from *Paralysis of the seventh pair*, resulting from ex-

¹ *I. M. G.* for April 1885, p. 15.

posure. The loss of contractile power of the orbicularis palpebrarum led to conjunctivitis, but the cornea escaped while he was with me. On a heatstroke-day, just after I had joined a new station, a native gentleman, born a Brahmin, residing two miles off, sent to me requesting that I would immediately visit his wife who 'was attacked with disease of the eye.' I went at once, and told her that she had a *Stye*. She replied that she was perfectly well aware of the fact, and gave me the Bengali name of the disease—which, perhaps out of spite, I have always failed to remember.

In the work quoted above, Sir Joseph Fayrer has given a very instructive case of destruction of the eye-ball and impetiginous eruption of the face in a Hindoo goldsmith. The lesions were attributed to peripheral irritation of the fifth nerve excited by a spark which flew into the external corner of his eye.

Valuable contributions to our knowledge of *Herpes Zoster Ophthalmicus* have been made by Dr. H. Garden¹ and by Mr. Scriven.²

Dr. Garden says that, of six cases in his list, the nasal branch was implicated in two: in one the eye was affected, in the other it was not. Of the remaining four cases, in which the eruption did not extend to the nose, the eye was also affected in one. In both these cases, the patients were old and debilitated. In the first, there was iritis, and in the other conjunctivitis. Hence Dr. Garden infers that Hutchinson's law,³ standing as it does, is far too absolute; for not only does inflammation of the eye, &c., occur frequently when the nasal branch is not affected, but also when the first division of the fifth itself is untouched; though it may be accepted as a fair inference that the chance of serious damage to the eye is greater when the nasal nerve is affected than when it is not.

Mr. Scriven gives the case of a Hindu, aged 40, treated in his Medical School Hospital, Lahore, the only example of this affection which he had seen in India. At first, there was periodic fever, occurring at night only. It was followed by severe left frontal headache, and, after a time, small vesicles appeared in clusters, at first on the forehead only, but, within two or three hours, on the side of the face also. There were two or three spots on the lower lip. On the fourth day of observation, there was a good deal of swelling of the left upper eyelid and slight inflammation of the conjunctiva with chemosis. On the following day, there was a small phlyctenula at the upper and outer margin of the cornea, and a little opacity near it. He was discharged on the twenty-first day, the report being: 'Cornea almost clear, vision perfectly good, no pain, slight vascularity of the conjunc-

¹ Reprint from the *I. M. G.*, 1874.

² *Ibid.*, *I. M. G.*, vol. iv. p. 139.

³ 'The eye scarcely ever suffers much when the nose is not affected, and the severity of the eruption on the one part is in direct relation with the severity of the inflammation on the other.'

tiva.' Mr. Scriven remarks that the case afforded an additional instance of the implication of the superior maxillary nerve. It also illustrated Hutchinson's observation that the side of the nose may show vesicles to its tip, and yet the eye may be only transitorily inflamed. He noted it as a point which he had not seen mentioned, that the eruption may extend to the cutaneous branches of the third or inferior maxillary division of the fifth, and show itself on the lower lip.

Conjunctivitis Excited by Mechanical Irritation.—At Cairo, I was called to see a fellow-passenger said to have been attacked with Egyptian ophthalmia. When I reached his hotel, the conjunctival irritation having passed off, he was quite well and dining heartily. Leaving in absolute darkness, I got upon broken ground where extensive building was going on, and was in great peril. At Bombay, I was summoned in haste to see an officer's child with an inflamed eye. The mother appeared frantic with terror, and desired me to recommend the best oculist in the place. Merely finding a little conjunctival inflammation of an hour's standing, I said that to take the child through the town in the heat of the day would do more harm than good, and recommended that the eye should be closed by a pledget of raw cotton, and that the little fellow should be allowed to go to sleep. This was done, most reluctantly. His recovery before evening brought me an elaborate apology and more than due credit.

The *Ophthalmia of Egypt* has been attributed to dust and sun-glare. The disgusting appearance of natives whom I saw at Cairo, showed that absolute neglect of cleanliness must often contribute largely to the causation of blindness.¹ These excitants are only too prevalent in the Delta of the Ganges; but, there, the great atmospheric humidity probably exerts a counter-influence. At the station of Bellary (Madras Presidency), the marked prevalence of ophthalmia among the European troops was attributed² to the extreme dryness of the atmosphere, to

¹ Dr. Colvin Smith mentions, in his *Report of the Expeditionary Force in Egypt in 1882*, that such affections of the eye occurred in 58·36 per 1,000 of European, and only 20·70 of native soldiers. Debilitated men were chiefly attacked. The severest type was, however, amongst the latter. 'Dirt' was, no doubt, a very common exciting cause, and it was considered that the Europeans were not always sufficiently careful in their ablutions, several using the same water in washing their faces, thus spreading the disease. The natives did not, of course, use basins. There were only thirteen cases of ophthalmia in three regiments of Indian cavalry. They were stationed nearer the desert and in a cleaner spot than the other natives. A piece of mosquito net thrown over the head to keep the flies off, and carbolic spray to drive them out of the rooms, were most useful. Five to ten grains of nitrate of silver to an ounce of water, and lotions of alum, belladonna, borax, &c., succeeded. No fighting man's sight was permanently injured, but one Kahar was reported to have lost his sight.

² *Madras Topographical Reports, Ceded Districts*, p. 76.

the excessive glare and reflection from the numerous large masses of granite in the lower fort, the light colour of the ground and numerous white-washed buildings, and to the fine white sandy nature of the soil. The disease commonly appeared as a mild purulent ophthalmia, but, occasionally, the deeper structures of the eye were involved and the cornea became opaque. Rarely, the organ became disorganised. Nitrate of silver and sulphate of copper were the usual applications.

In Paulghaut the 'country sore eye' was noticed as being very prevalent in April and May, amongst Europeans as well as natives. In Dindigal, ophthalmia occurred annually, as an epidemic, during July and August, especially among children, the conjunctiva becoming highly inflamed with tumefaction of the eye-lids, and a purulent discharge. It was generally attributed to the effects of the hot winds. The best application was considered to be solution of lunar caustic.¹

In his 'Medical Memoir on the Plains of the Indus,'² Dr. Perceval Lord observed that the sandy and saline deserts towards the south at once suggest the idea of ophthalmic affections, and he found them so common that he scarce met a man whose eyes were not more or less inflamed. At Khaispin, where he saw on an average a hundred patients a day, he examined more cases of pterygium, opacity of cornea, obstructed lacrimal gland, and other external affections of the eye, during the week he remained there, than a London hospital would present in several months.

In his valuable account of the 'General and Medical Topography of Ajmeer,' Dr. Robert Hamilton Irvine spoke of ophthalmia as very severe and common in the province. He considered that it is caused by the 'hard particles of dust impinging on the eyes during the frequent high winds.' He did not regard it as being epidemic or contagious. It was very amenable to treatment, but the natives frequently aggravated it into a severe lingering complaint.

In his report on the diseases of Ladak, Dr. Cayley notices that conjunctivitis is very common there, hardly ever appearing in the acute purulent form; but being generally of a very chronic character, the result of a low form of muco-purulent inflammation of the conjunctiva, and lasting weeks, months, and even years, leading to great thickening and cedema of the lids, and extreme vascularity and roughness of their mucous membrane, causing opacity of the cornea, and often producing entropion, of which affection he cured several cases by operation. He found that treatment by astringent lotions, the application of sulphate of copper to the inner surface of the lids, and nitrate of silver painted on outside, was often effectual; but, in many cases, the disease was too confirmed for rapid amendment. He attributed many of the cases to exposure to the intense glare and dust, and to the

¹ *Madras Topographical Reports* (Southern Division), p. 143.

² *C. M. T.*, vol. viii. part 2, Appendix.

heat of the sun in the barren sandy deserts which extend over much of Ladak.

In the 'Madras Medical Journal,' vol. iv., p. 333, Mr. Bucke published a report, which may still be consulted with advantage, on ophthalmia, as it existed, in 1834, among the boys at the Military Orphan Asylum, Madras. Five hundred and seventy-two cases were admitted in eighteen months. The disease appears generally to have commenced as mild conjunctivitis; but, many of the boys being in weak health, there was a tendency to sloughing of the cornea. There was sometimes purulent ophthalmia with granular lids, at others pustular conjunctivitis. Corneal ulcers 'were often factitious, from the application of *chunam*' (lime) 'or caustic.'

It was reported, in 1842, that improvement in the ventilation and cleanliness of this asylum, the inmates of which were mostly Indo-Britons, had, in the two years ending 1838, reduced the cases of ophthalmia to 293, the admissions, during the two preceding years, having amounted to 1,057. Here we find a clue to the origin of most of the conjunctivitis which prevailed in military buildings for Europeans in old times.

Conjunctivitis used to cause a great deal of suffering among the European men, women, and children cooped up in the ill-ventilated and often over-crowded barracks which existed before my time in the North-West Provinces of India. Perhaps those who attributed this to the practice of lime-washing the floors were partly in the right. It was a well-known trick of the regimental urchins to irritate their eyes that they might avoid going to school. Confinement in a cell containing a heap of quicklime has been mentioned as a torture practised in India.

Morehead observed that measles in the Byculla schools were generally followed by troublesome catarrhal ophthalmia.

Mr. Macnamara speaks of muco-purulent conjunctivitis occasioned by an insect finding its way into the eye. There is especially one insect, 'the flying bug,' which appears to be gifted with a large share of the venom of the *mylabris cichorei*, or blistering fly of India, the contact of which produces a sensation like a knife-stroke through the eye, and a good deal of subsequent conjunctivitis with œdema of the lids. As it is black and large, it is easily removed.

There is still a great deal of 'ophthalmia' in the armies of India. Bryden's tables show that, in ten years, there were admitted 17,561 men of whom 569 were invalided; 2,181 women and 7,221 children. No deaths.

A medical officer in Fort William lost the sight of one eye, with considerable deformity, in opening a bottle of liquor ammoniæ. In very hot weather, a lady had fainted in a room near his dispensary. In his haste, he did not take care to hold the bottle, the stopper of which had stuck, at arm's length; there was an explosion straight in

his face. A similar accident, attended by far less serious results, occurred to my late friend Joseph R. Bedford.¹ Whilst on his passage to India, he was endeavouring to remove the glass stopper from a bottle of muriatic acid. It resisted, he applied force, holding the bottle incautiously too near his face; on a sudden, the stopper was removed and the fumes rushed out. He experienced a sharp pain in both eyes, and made up his mind for life-blindness. Sweet oil was at once applied; and, on opening his eyes in about a quarter of an hour, he found, to his intense joy, that vision was unharmed. A slight conjunctivitis continued some days. I do not think that I have ever been more shocked than I was to see an old gentleman apply a strong Goulard lotion, mistaken for a zinc wash, to both his eyes. At first, the pain and redness were intense, but they soon abated under warm water sponging. No lesion followed.

I know of more than one case in which surgeons have been blinded by venereal matter in opening buboes, &c. Mr. J. D. O. Wild reported² the case of a Bengal hospital apprentice of irregular habits who, six days before coming under treatment, contracted inflammation of both eyes, which he attributed to the accidental introduction of venereal matter while dressing sores. There was severe purulent ophthalmia of both eyes. Right much distended, its structure disorganised, sight gone. Left inflamed and swelled, but with a clear cornea. There was a tendency to delirium and coma, but, on being roused, he spoke coherently. Previous to the attack, he was suffering from quotidian fever. He died in fifteen days with symptoms of meningitis. The *right* eye was totally disorganised. Meninges red and congested. A large patch of 'ulceration' coated with pus occupied the surface of the *left* hemisphere.

My friend, Dr. Samuel Brown, showed me, before I went to India, many cases of *Pustular Conjunctivitis*, as it used to prevail among unhealthy-looking factory girls at Belfast. He used to cure it by a dexterous application of the stick of nitrate of silver. I never saw it in London or in Bengal, and Macnamara does not say that it occurs in India, but it used, as I have stated above, to prevail in the Madras Orphan Asylum. My friend Dr. Henry Cayley, in charge of the Calcutta Eye Infirmary, informs me that the 'strumous' ophthalmia of children is not uncommon among natives and Eurasians in India. He thinks he has seen it in European children also, but of this he is not quite sure, as he has not seen it, among them, in its most characteristic stage. European children in India are not generally of the class that are most liable to that form of ophthalmia. Judging from hospital experience, his impression is that scrofulous ophthalmia is much more common in large English cities than in Calcutta.

¹ 'Chemical Injuries to the Eye,' Finch's *I. M. J.*, vol. ii. p. 481.

² . *A.*, No. 8, p. 661.

I watched very marked *Leptous Conjunctival Deposit* in an East Indian woman for some weeks, and in a *mheter* (sweeper) for many years. I could not perceive that it made any progress in either case.

Pterygium, in every one of its forms, is very common among the natives of India. Mr. Brudenell Carter observes¹ that 'pterygium is especially apt to occur in persons who have lived in the tropics.'

In a report on the medical topography of Orissa² Dr. Shortt mentions that pterygium is so very common throughout that province that, in almost every village, he met with two, three, or more cases. The growth extends over the cornea, producing blindness. In several cases, he dissected away the corneal portion. A few drops of the solution of argenti nitras for a day or two completed the cure.

The number of pterygia successfully operated upon, in four years, by my late friend Mr. Martin, at the Calcutta Eye Infirmary, was 58 in 44 individuals. I saw *Epithelioma* of the conjunctiva, beginning at the outer canthus in an English officer. At a meeting of the Calcutta Medical Society, held in December 1880, a case of Dr. Cayley's was brought forward in which a growth, considered to be papillomatous, originated, like a pterygium, at the outer canthus. This, after ablation, re-formed, spread, and extended over nearly the whole of the cornea, abolishing vision. The eye was removed, the growth was confined to the sclerotic and cornea, not penetrating the eyeball.

Diseases of the Cornea.—In 1858, my friend Dr. W., coming home one afternoon from a long round of visits in Calcutta, sat upon the edge of his low bed and opened a bottle of soda water. Slipping from his fingers, the cork flew upwards and struck his cornea with its angular outer margin, producing a most painful shock and denting in the cornea as if the edge of the thumb-nail had been strongly pressed vertically across the axis of vision. It appeared as if the superficial layers of the cornea yielding, the inner ones had been fractured. The sight of that eye was lost; the dent remained visible for years, a small opacity occupying the axis of vision.³

Mr. Macnamara gives an interesting case of a Hindoo boy with characteristic malformation of the incisors, suffering from congenital syphilis and *Keratitis* of both eyes.

Extremely marked *Arcus senilis* frequently occurs in India. I was consulted in the case of an English gentleman in whom this condition was regarded as a bar to life insurance. A very active and healthy-looking Scotch gentleman had, in middle age, a singularly pronounced arcus. He lived for about fifteen years after his return

¹ *Holmes's Surgery*, third edition, p. 705.

² *I. A.* No. 10, p. 508.

³ A few years ago, it was reported that a man was struck in the eye by a ginger-beer cork; the blow did not cause much pain at first, but, after a time, he began to suffer so much in the eye that he was admitted to the Bristol Infirmary, where he died from 'cerebral congestion.'

from India. A very remarkable case was brought into my ward and taken out again so quickly that I only saw him once. He was a respectable European who, although not much over 50 years of age, had an aspect of extreme senility. He had a good deal of the look of an albino, but I think that he had been a very fair red-headed man. His hair was snowy white and he had the arcus excessively developed. His feet were gangrenous; but, in the legs, the anterior tibial muscles only appeared to have mortified up to their origins, showing lividly through the pallid integument.

I have already described *Sloughing of the Cornea*, as it occurs most extensively among the native poor in Bengal, as a form of malarial cachexia (*vide* p. 256). Undoubtedly, scorbutus is, in many cases, a factor in the causation of this tendency to sloughing of the cornea. I have already alluded to the fact that this death of the cornea precisely resembles the state produced by Majendie in dogs, by dividing the fifth nerve or by feeding them exclusively upon non-nitrogenous food. In an excess mortality in a native jail, attributed to a diminution in the dietary, which occurred in 1853, my friend, the late Mr. Bedford, observed¹ cases in which the corneæ ulcerated and sloughed away previous to death. In the chapter on scorbutus (p. 351) I have cited the narrative of my friend, Mr. Alexander Grant, to the effect that, in the expedition of 1840 to China, the 37th Madras Native Infantry, being ill-fed and miserably lodged, became thoroughly scorbutic; and then sloughing of the corneæ was rife among them.

In Lower Bengal, this destructive corneitis occurs frequently in patients worn out by jail diarrhoea, perishing from sheer inanition, often without any very manifest evidences of a scorbutic taint, although the presence of such a condition may generally be expected (*vide* p. 256). When the Andamans were first occupied as a place of transportation for criminals engaged in the Great Mutiny, the malarious influence was intense and most destructive; and, nearly all food having to be brought in ships from Calcutta, scurvy became rife among the prisoners at Port Blair. Dr. Gamack observed, 'Sixteen deaths are recorded amongst the patients admitted under Diseases of the Eye. These occurred from scurvy or diarrhoea, and it frequently happened that the scurvy patients became blind some time before death. A patient, in the advanced stage, would complain of violent pain in and around the eye; and, on examination, nothing could be detected but a slight haziness of the cornea; but inflammation would proceed so rapidly that, in 24 or 36 hours, the globe of the eye would become filled with pus; and, if both eyes were not affected at first, the other was almost sure to become affected after a few days. The sign was looked upon as fatal' (it is too often so in Bengal); 'but two cases which occurred towards the end of

¹ *J. A.*, No. 2, p. 688.

the monsoon are still alive, and the men, although quite blind, are now well and have become exceedingly stout.'

In those who survive, all the results of penetrating ulcer of the cornea, *anterior synechia*, *prolapsus of iris*, *staphyloma*, &c., may occur. Mr. Macnamara recommends iridectomy in spreading eccentric ulcers of the cornea.

Mr. William Martin operated, at the Calcutta Eye Infirmary, upon fourteen cases of *Staphyloma* in four years—not a large number considering how great is the prevalence of ulceration of the cornea in Bengal.

Syphilitic and Rheumatic Iritis occur in India nearly as in Europe. Dr. Robert Bird relates ¹ the very noteworthy case of a European in whom daily attacks of feverishness with pains in various parts of the body, lasting from two to three hours, was followed by conjunctivitis and characteristically marked iritis of one eye. Being in a weak state of health and not improving under treatment, he went on a sea-voyage to the Straits, and returned to Calcutta, in five weeks, as ill as when he started. He was very lean and weak, and, to all appearance, the sight of the eye was destroyed. Suddenly, he was seized in the middle of the night with fever and ague, and, from this time, he steadily mended. Pain and morbid sensibility left the eye, which gradually recovered.

I was shown a patient who, having had solution of atropia applied to the eye, had marked symptoms of belladonna poisoning, and I know of two instances in which native patients, under the same influence, made their way out of the windows of an upper ward, but happily were not killed.

When I first went to India, I found *Chronic Glaucoma*, confined to the crystalline, in its first stage, so very common among natives of Bengal beyond middle age, that I was in the habit of looking for it in men who did not complain of eye disease. The form of glaucoma most frequent among natives appears to be this first stage, which Mackenzie used to describe as often remaining stationary for a great part of life. Mr. Odevaine has recorded some valuable remarks, in the 'Indian Medical Gazette' for July 1877, on the importance of making an accurate diagnosis between glaucoma and cataract.

Surgeon-Major Drake-Brockman, Superintendent of the Madras Eye Infirmary, has given ² an elaborate analysis of the cases of 73 persons (108 eyes) suffering from glaucoma. The greatest benefit was derived from the instillation of eserine with the removal of a segment of the iris. Of the 41 who were so treated, 13 recovered with vision. He expects that eserine with sclerotomy (which latter gave 4 recoveries in 9 cases) are likely to afford the most favourable results.

Mr. Macnamara remarks, after giving illustrations of the syphilitic form, that non-syphilitic opacity of the vitreous commonly arises

¹ *I. M. G.*, vol. i. p. 56.

² *I. M. G.* for October, 1885.

(excluding cases depending on disease of the choroid) from an impoverished state of the blood induced by malarious influences.

My late colleague, Mr. Cutcliffe,¹ described the case of an East Indian, *æt.* 55, who was treated in our hospital for *Exophthalmos* of both eyes due to syphilitic disease of the base of the skull. He died convulsed.²

Malignant Diseases of the Eyeball.—Many specimens of various forms are preserved in the Calcutta Medical College Museum :—252, ‘epithelioma of the ball of the eye, springing by a broad base from the sclerotic adjacent to the cornea’; 293, ‘An encephaloid tumour between the ball of the eye and the orbit, pressing upon and causing flattening of the optic nerve prior to its penetration of the sclerotic’; 294, glioma, fungating; Preps. 283, 295, 306, 315 and 317, represent medullary cancer of the eye; 320, ‘A very unique specimen of melanosis of the ball of the eye, causing complete disorganisation of the organ. The section is now of a chocolate colour.’

Dr. Robert Brown has reported the case of a Mussulman, of Sylhet, aged 20, with melanosis of the eyeball. The organ was enucleated.³

Mr. C. Macnamara says that he considers Cancer of the orbit to be common. Fresh cases of this formidable disease were seen every year in the Calcutta Hospital, whereas the other forms of malignant disease were rarely seen. Mr. Macnamara’s predecessor, Mr. Martin, had only three cases of excision of the eyeball, for malignant disease, in four years.

Considering the unusual prevalence of enervating influences in India, it is remarkable that a larger number of cases of *Amaurosis* should not be observed there, among Europeans and natives.

In an old report of the Madras Eye Infirmary, it is stated that the majority of 877 cases of amaurosis (only 12 of which were Europeans) were treated for partial loss of sight, ‘depending chiefly upon exposure to the sun and night chills;’ many instances were ‘the result of febrile disease and inanition succeeding parturition.’

I, for some years, watched with interest the condition of a well-grown and healthy English gentleman, who was rendered quite incapable of work in India, by what appeared to be mere congenital weakness of vision. There was no visible defect in the eyes, and the case was not one of amaurosis. He retired from India soon after the age of 30.

In the old report of the Madras Eye Infirmary cited above, it is mentioned that *Nyctalopia* is a very prevalent disease in that country;

¹ *I. M. G.*, vol. vii. p. 276.

² *Vide* Mr. Hutchinson’s case of proptosis in an Indian patient of mixed race, *M. T.*, vol. ii. for 1884, p. 59.

³ *I. A.*, No. 15, p. 272.

672 cases, of which only 4 were in European soldiers and 8 in sepoys, having been treated in nine years. It was found to be principally confined to the native agriculturists and artisans, and was seldom met with in the European and the Indo-Briton. The alleged causes were exposure to the strong dazzling light and ardent heat of a tropical sun by day, and to chilling dews by night, severe attacks of fever, scanty and bad food, 'a disordered state of the alvine secretions,' and worms. Females were frequently observed to be affected with this complaint after parturition. It was considered that many of the cases of amaurosis and cataract treated in the infirmary had their commencement in night-blindness.

Nyctalopia used to be very prevalent in native jails, probably as an expression of scorbutic cachexia.

While I was a member of the medical committee for the Calcutta Pilot Service, I saw a great deal of *Hemeralopia* in men who attributed it to the incessant movement of the sun-lit sea when they were stationed in the small light-vessel moored at the Sand-Heads. Weavers are liable to be similarly affected by the constant tremulous movements of the threads.

Dr. J. W. Moore concludes some valuable remarks¹ on nyctalopia and hemeralopia, as they occur in India, with the following summary of treatment which represents the practice usually followed in that country:—'Attention to the general health and prevention of exposure to the exciting causes. Blisters behind the ears, or on the temples have been recommended, but are rarely beneficial. A similar remark applies to leeches. Tonics are always required. But the most important means of cure is confinement during the day in a darkened room. I have found this plan perfectly successful within a week, and it is highly recommended by others. Exercise should be taken in the dark, and companionship provided for the patient to as great an extent as possible.'

I always gave quinine in Bengal. An antiscorbutic course will be needed in a large proportion of cases; and, even where signs of scurvy cannot be detected, it will never be wrong to give the patient the benefit of acting on the suspicion that he may be the subject of a scorbutic taint.

Snow Blindness.—A friend and brother officer of mine, who was an excellent landscape painter, greatly injured his eyesight by too anxious endeavours to represent the outlines of the snowy range from Khatmandoo. The degree of his blindness fluctuated; but, although much consideration was shown to him, he was never practically fit for duty. He died early in England, as I understood from brain-disease. Dr. Cayley has noted² that ophthalmia appeared, in many cases which

¹ 'On Maladies Attributed to Lunar Influence, &c.,' *I. M. G.*, vol. iv. p. 181.

² *I. M. G.*, vol. iii. p. 4, 'Notes on Ladak.'

he treated among the inhabitants of Ladak, to have arisen from the glare of the snow whilst crossing mountain-passes, as a protection from which the natives often wear snow-spectacles made of plaited hair.

Muscae Volitantes, generally due to functional causes, are frequently complained of in India. Twenty-three years ago, I was sitting writing in a room which was shaded, except that the sun-light entered rather more freely than I usually allowed it to do, and fell upon my left eye sideways. Suddenly, there appeared a vivid play of sharp iridescent lines, as if quite outside the temple on that side, rapidly moving in zigzags, like the lines of an old-fashioned fortification. This has occurred on one or two subsequent occasions, but never since my return to England, more than nine years ago, until, sitting with the left eye towards a moderate light, in cool rainy weather, in July 1884, the lines appeared in front and on the outer side of that eye. The lines were, at first, of hair-like tenuity, but brightly coloured. In a few minutes, they formed a fortification-like line of prismatic colours, in which blue prevailed, about a sixth of an inch broad. There was not any headache or other uneasiness. On closing the left eye and then both eyes, they did not disappear. The appearance passed off in about a quarter of an hour. A healthy brother officer complained to me, in India, of the same symptom, which caused no more uneasiness than the flash of a lucifer match would do. This did not appear to point to any ill-consequence in either case. This play of colours has been repeatedly noticed by observers at home.

The first observation of *Filaria Oculi* is ascribed by Mr. Curling, in his lectures on morbid anatomy, to Dr. Nordmann, of Odessa. In the sixteenth number of the 'Indian Annals,' Mr. Charles Macnamara gave the details of two cases of filaria papillosa in the anterior chamber of the human eye, *vide* p. 330 of his manual. In the first volume of the 'Transactions of the Medical Society of Calcutta,' are elaborate papers, by Messrs. P. Breton and Twining, on 'Worms found in the Eye of the Horse.' Mr. Brett also made some remarks on this subject.¹ Much more recently, my friend, Dr. W. B. Beatson, now of Bath, has published² a valuable account of filaria as infesting the eye of the horse in India.³

A vast amount of *Cataract* occurs in India. The 'Statement of Moral and Material Progress and Condition of India during 1881-2' shows that, in the twelve months, cataract was operated upon in 2,801 persons, 1,803 of whom were restored to good sight.

As is well known, Mr. Macnamara has long practised, with great success, a modified operation for linear extraction without iridectomy.

¹ *I. M. J.*, N. S., vol. v. p. 493.

² *I. M. G.*, vol. i.

³ In cases of worms moving under the conjunctiva in negroes, cited in *I. M. J.*, vol. v. N. S., p. 179.

During the five years preceding the publication of his work in 1868, he had operated, in India, on upwards of six hundred cases, the results surpassing those which he had obtained by any other operation for the removal of an opaque lens. He considers the operation to be equally applicable to cases of either hard or cortical cataract. He points out that, after this mode of extraction, we have much less cause for anxiety as to the occurrence of that most terrible complication of flap extraction—suppurative keratitis. He adds: 'Even among the poor rice-fed Hindoos, who form a considerable portion of our hospital patients in Calcutta, we seldom meet with instances of suppuration of the cornea following linear extraction. I hardly exaggerate the case when I assert that some thirty per cent. of the operations performed in Calcutta formerly failed in this way.'

The native operations, by reclination and solution, have been described by several authors. Mr. Breton¹ saw Sautcouree, a Mussulman, operate in Calcutta. His narrative is fully illustrated by plates. Mr. Raleigh also described the native operation.² Dr. Percival Lord gave an account of the operation which he saw employed by a native at Dera Ismael Khan.³ He considered that the operation ended far more frequently in the total destruction of the globe than in the restoration of even an imperfect vision. Mr. Macnamara stated that hardly a week passed in which he did not see, in the Calcutta Hospital, several of the huckeems' and kobirajes' patients suffering from either inflammation of the choroid or from retino-choroiditis. In 1876, Dr. Cayley successfully extracted an opaque lens in a leper.⁴

Recollecting how frequently *Strabismus* was met with in England before the practice of tenotomy was adopted, and having travelled a good deal in out-of-the-way parts of India, I think it certain that the natives of India are far less subject to squinting than the inhabitants of Europe are. Several of my retired brother officers, of great up-country experience, have made the same remark. Dr. John Murray does not quite agree with us. He tells me that, when the operation was first introduced, he, in driving through the streets, used, whenever he saw a man who squinted, to tell him to jump up upon his vehicle, and to take him to the dispensary, where he generally had an operation every day. *Strabismus* occurs in Hindoos, Mohammedans, and Eurasians; and I have noticed that European children, born in India, are very liable to acquire it on coming to England.

Two ladies, born in India, have large families, also born in India. Four of these children squinted. The parents and grandparents are free from visual obliquity.

(For *Exophthalmic Goitre*, see 'Bronchocele,' page 365.)

¹ *C. M. T.*, vol. ii. p. 341.

² *Ibid.* vol. vi. p. 137.

³ *I. M. J.*, vol. iii. p. 402.

⁴ *I. M. G.*, April 1876.

Congenital Defects.

Cyclops.—Prep. 1010 in the Calcutta Medical College Museum is 'a monstrous fetal calf. There is only one ocular socket containing the eyeball, which is provided with two corneæ, and protected by imperfectly developed upper and lower eyelids. The eye is in the centre of the forehead (cyclopean).'

Mr. G. C. Hall reported in the 'Indian Medical Gazette' a case of *Dermoid Tumour of the Cornea and Sclerotic* in a native. It was of the same brown colour as the skin of the face, and had three long hairs growing out of it. He dissected it off and the patient got perfectly well, the cicatrix contracting to about half. Microscopical examination showed that it consisted of true skin with subcutaneous fat, sweat glands, hair bulbs, and everything complete. Assistant-Surgeon R. K. Mahomed¹ removed a dermoid tumour from the conjunctiva. The part healed well.

A poor little native *Albino* boy, suffering from malignant disease, in one of our surgical wards, had the characteristic ocular defect very markedly. Although his bed was as much as possible in the shade, daylight greatly depressed and embarrassed him; but, towards night-fall, he became playful. I have happened to see the *Tapetum choroideæ* twice, once in a young lady of very light complexion, but by no means an albino; the other time, in a young man with dark brown eyes and hair. In the first, the tapetum shone out, in a darkened room, brassy yellow; in the second, it had a deep ruby lustre.

DISEASES OF THE NOSE.

The history of *Rhinoplasty* is too well known to need recapitulation here. It was adopted by the huckeems to repair the mutilations which were long commonly practised and are not yet quite disused in India, and of which I have given an account at p. 487 of my 'Medical Jurisprudence.'

For *Lupus* and *Rodent Ulcer* see pp. 357 and 359.

Ozæna—*Peenash*.—In various hot climates, maggots have been noticed to infest the noses of dying animals. In 1868, Dr. N. Jackson, of Deega, near Patna, sent to the editor of the 'Indian Medical Gazette'² larvæ found at the summit of the nostrils of full-grown sheep. It is not mentioned under what circumstances the animals died. They were examined by the late Dr. Stolicza, and pronounced to be a species of a large cestrus gadfly. He stated that these creatures lay their eggs in the nostrils of various ruminants, and that the young larva, when developed, walks up the nasal bones and ensconces

¹ *I. M. G.*, January 1879.

² Vol. iii. p. 278.

itself in any part of the surrounding tissue. It frequently lodges itself in the brain. These larvæ are very common in the heads of sheep in the Punjab. I am told that a like condition is frequent in England.

In 1827, Mr. W. L. MacGregor reported¹ the case of a man, suffering from venereal disease of the nasal bones, in which larvæ, fully half an inch long, were discharged from the nose alive and active. The pain, which was excessive, occupied the position of the antrum, and he became almost delirious. The means employed for his relief were an injection of turpentine and laudanum in olive oil, the vapours of Aq. Ammonia and sulphuric ether, and an infusion of tobacco gr. x to the pint. Under this treatment, he expressed himself perfectly recovered on the fifth day. The number of living maggots discharged was upwards of a hundred. When he was at the worst, he was '*literally covered with flies.*'

In 1856, Dr. Dempster placed on record² the case of a man who suffered two attacks within thirteen months, in which upwards of 300 and 172 larvæ of the blue flesh-fly came away. During both attacks, he was *much teased with numerous blue flies*, buzzing about his head and face.

Dr. Dempster's report embodies a valuable abstract of the literature of this subject, giving the observations of Morgagni and Boerhaave, and facts relative to the presence of the larvæ of the gad-fly in the nasal passages in the West Indies.

In the 'Bulletin Médicale Belge,' 1839, there is notice of the case of a woman in whom the entrance of a fly to the nostril was followed by severe frontal pain, often so distressing as to cause delirium. Several full-formed grubs were discharged. Upwards of fifty came away during the next week. They were those of the common fly; several, being kept under favourable circumstances, became flies.

Baboo Taruck Chandra Lahory has given³ a very careful description of this state, under its vernacular name of *Peenash*, a word of Sanscrit origin signifying disease of the nose. He relates five cases, in at least three of which it appears probable that syphilitic disease existed. He says that, in five years, there were 91 admissions at the Allyghur Dispensary for worms in the nose; of these, 46 recovered, 14 were relieved, 2 died, and 29 ceased to attend. In a fatal case, he found 'worms in the cribriform plate of the ethmoid bone, ulceration' (?) 'of the bodies of the sphenoid and ethmoid bones, and slight congestion of the vessels of the brain.'

Dr. D. T. Lyons has recorded⁴ an important contribution to the literature of this subject under the title of 'An Account of a Disease of the Nose and Cranial Sinuses prevalent in the Zillah Rhotuck, in

¹ *C. M. T.*, vol. iv. p. 28.

² *I. M. J.*, N. S., vol. i. p. 45.

³ *I. A.*

⁴ *Ibid.* No. xv. p. 45.

the Punjab.' He says, 'Invariably there is a depression or flattening of the bridge of the nose.' There was not in two out of Lahory's five cases; but, doubtless, this is merely a question of degree.

This disease, or rather complication of disease, is again described by Dr. W. J. Moore in his account of Marwar.¹ He says, 'as a rule, persons so afflicted will be found to have suffered from syphilitic ulceration. In some instances, however, the primary condition is scrofulous inflammation, or other affection of the nasal mucous membrane. This affection is frequently seen in the nose of the camel. Three valuable contributions to this subject will be found in the fifth volume of the 'Indian Medical Gazette.'² Dr. Alexander Garden shows that, in four years, 303 cases of ozæna were treated at the Saharunpore Dispensary. Out of 168 cases which were sifted, peenash occurred in 63. Ozæna was found to be more frequent among females than among males; the persons being, as a rule, the very poor, weak, emaciated, half-starved anæmiated sufferers from fever, exposed to vicissitudes, and living a precarious life, the day's meagre meal most often depending on the day's wage. No marked connection has been traced between this and malarial and other diseases of the liver and spleen, though such might be supposed to exist from the frequency with which epistaxis is met with as the first symptom. In some eight or ten cases, there was an undoubted syphilitic taint. Dr. Garden shows that ozæna is rare before puberty, but is common afterwards. It occurs chiefly in the hot months of the year. It ends, as a rule, in recovery, often leads to injuries of the soft parts, occasionally ends in death. It is essentially a disease of debility, without any marked connection with scrofula.

In a patient treated by Dr. Higginson, 56 maggots were discharged, all living, from the nose. 'These were put into a bottle, over the mouth of which a piece of paper was tied. They went through the necessary changes, and all developed into able-bodied blue-bottle flies.'

Dr. Whylock³ treated a European soldier, aged 24, the subject of secondary syphilis, who suffered in this manner. The maggots became blue-bottle flies. Turpentine vapour was very successful in this case. Baboo Bhawani Doss⁴ values chloroform in these cases.

Dr. Thomas Mayne relates⁵ the fatal case of a man with old (syphilitic?) perforation of the soft palate, whose nasal passages were the resort of live specimens of *cæstrus hominis*, varying in size from four to six lines.

For a notice of a similar affection of the auditory passage, see the the chapter on Diseases of the Ear.

For *Nakra*, an acute inflammatory thickening of the Schneiderian

¹ *Ibid.* No. xx. p. 524, and *I. M. G.* for August 1871.

² Pages 38, 106, and 172.

³ *I. M. G.*, October 1877, p. 263.

⁴ *Ibid.* for July 1876.

⁵ *Ibid.* January 1875.

membrane, to which natives of Bengal suffering from fever are liable, see 'Malarial Cachexia' (p. 270). I do not think that, at least in Bengal Proper, *nakra* is liable to end in *ozæna*. It is, however, allowable to argue that the *ozæna* of Upper India is an aggravated form of *nakra*. Both occur in sufferers from malarial fever. The following is Dr. Garden's description of the symptoms and progress of *ozæna*—apart from the miserable complications of syphilis and *peenash*. In a few cases, *coryza* was given as the starting point. The first stage is essentially inflammation of the Schneiderian membrane; low in type and rapidly tending to ulceration. In many, if not most, the first sign is a sense of fulness and discomfort in the nose, accompanied by more or less pain, and soon followed by a thin foetid ichorous discharge. In some, the foul odour is perceived for some time before there is any discharge. At this time, the mucous membrane will be found in a greater or less degree inflamed, dark red, and swollen. Very frequently, the first symptom that attracts attention is epistaxis; and the case, if seen at once, may never go beyond this point, and, hence, figure in the returns as epistaxis. Dr. Garden has met with numerous cases in which, after a time, the diagnosis has had to be altered in accordance with facts. In 1869, of 75 cases, 22 commenced with bleeding from the nose; and, in most of 8 others, entered as epistaxis, it is very questionable whether the true ultimate diagnosis would not have been *ozæna*, as the epistaxis had been going on in some cases daily for weeks. In all cases where the first symptom was epistaxis, the discharge became continuous, at first apparently nearly pure blood, then sanious and foetid, then merely tinged with blood, and, finally, more or less yellow in colour, the foul odour remaining the same.

Whatever the first symptom, the discharge, after a time, becomes thin, purulent, or muco-purulent, sanious, ichorous, and foetid. After a longer or shorter interval, if the case is not treated, ulceration of the mucous membrane occurs. In the majority of cases, especially when proper treatment has been employed, the disease goes no further. The discharge is less purulent, sanious, and offensive. The ulcers, chiefly superficial, heal, and the mucous membrane resumes its normal condition. In some few cases, the stage of ulceration is not attained. But, in a considerable proportion, no such favourable termination occurs. The ulceration is extensive, and involves structures deeper than the mucous membrane. From the first, the skin of the nose, and even of the face, presents an erysipelatous condition, is swollen, red, hot, and painful, whilst the surrounding parts are oedematous. The skin over the bridge of the nose ulcerates or sloughs, the nasal bones become carious or necrosed, the bridge of the nose is destroyed and a permanent deformity is left. Dr. Garden has seen a sinus left by the destruction of the soft parts of the nose below the nasal bones. The inflammation may extend to the antrum of Highmore, or even

originate there. One of Dr. Garden's earliest, and certainly most severe cases, commenced with epistaxis followed by the usual symptoms. The inflammation spread to the antrum and mouth, and, ultimately, to the contents of the orbit, through the nasal duct. Sloughing of all the implicated parts supervened, destroying them all, and the patient's intense suffering only ended with death.

The parts most commonly affected are the superior and middle meatus. In these cases, the inflammation is not unfrequently found extending to the palate and gums, and even to the soft palate, giving them a dusky red erysipelatous look. He has seen sloughing of the gums and perforation of the hard palate. In one case, there was marked pytalism. In nearly all, there is an erythematous condition of the skin of the upper lip, caused by the contact of the acrid discharge.

The amount of pain varies much, but pain is rarely absent. It is often referred, chiefly or entirely, to the vertex or forehead, being apparently sympathetic and affecting the frontal and other sensory nerves of the scalp. When the upper meatus and the frontal sinuses are involved, the pain is referred to the forehead, and is constant and agonising, destroying sleep night and day. In slight cases, and at first, there are no very marked constitutional symptoms. There is no fever; the tongue is, as a rule, furred, and there may be anorexia. Usually, there is a weak quick pulse, with considerable general ability. In the more severe cases, the constitution suffers to a considerable degree. Loss of rest and of appetite, diarrhœa, dysentery, hectic, are all met with the results of an exhausting disease, and possibly of poisoned blood.

Dr. K. P. Gupta treated a Ghoomkahi wood-cutter in whom epistaxis was found to result from the presence of two large *Leeches* in the nasal passages. He used turpentine. When leeches were much employed in Indian practice, an onion, instead of salt, was always used to make the creature drop off.

The following cases are cited by Dr. Gordon¹:—A naturalist, who had for some months travelled in Formosa, suffered from epistaxis and pain in the head. A leech was perceived in the nostril through a speculum, protruding its head between the blades of the instrument. Its head was seized with an artery forceps and salt and water injected. In a little time, it came away; it was an inch and a half long. He recollected to have drunk from a pool in which he noticed some leeches about half an inch in length, and he afterwards picked two or three such from the roof of his mouth.

A gentleman was affected in a similar manner. Every now and then, the head of the animal would be protruded from the nostril, and wander about over the lip and nose. Its owner could generally cause it

¹ *Epitome of Reports of Medical Officers of Chinese Customs Service*, p. 178.

to protrude by dipping his face in a basin of water. At Formosa, a monkey had a leech in its nostril.

For *Epistaxis*, see 'Malarial Cachexia' and 'Ozæna,' pp. 246 and 473. A remarkable case in which the death of a European soldier appears to have been caused by arterial hæmorrhage from the nose is related¹ by Mr. F. Sievwright. Two days previously, having been in a state of inebriety for several days, he fell down a flight of stone steps and injured the head and nose, marks of violence being evident on both. He lived two days and a night after the hæmorrhage commenced. No fracture of the base of the skull could be detected; liquid blood was effused between the skull and dura mater in the left occipital region, and blood clots were found in the frontal sinuses and in the antra. Death was not fully explained, but it was probably due to that hæmorrhagic tendency which is so frequent in systematic inebriates.

Preparations 189 to 195 in Ewart's Catalogue represent specimens of *Gelatiniform Nasal Polypi*.

In the case of an old Mahomedan, who had nasal polypi situated far back, Mr. B. Colaco² employed tannic acid snuffing for five days, as recommended by Mr. Bryant. The tumours 'could then be squeezed out of the nose piecemeal.'

In the four years ending 1883, the returns give under this heading 3 cases in Europeans; no admissions of women or children; native soldiers, 16; prisoners, 18.

There is a very fully reported case by Dr. R. Browne³ in which the death of a Northern European was caused, in India, by a nasal polypus, apparently non-malignant, which made its way upwards through the base of the cranium by partially destroying the ethmoid and sphenoid bones, occasioning meningitis.

Prep. 78, Series 16, in Mr. Sibthorpe's catalogue of the Madras Museum is a fibrous nasal polypus attached to the base of the skull, from which it had apparently grown. It had been noticed for a year. 'Removed from a native male, æt. 18, who died from shock.'

Rhinoliths.—Assistant-Surgeon Bhowanee Day⁴ found the nostril of a Mohammedan woman, æt. 35, who was suffering from profuse epistaxis, distended by a protruding stone. When removed, the rhinolith was found to present a perfect mould of the nostril. It was one-and-a-quarter inch long and three quarters of an inch broad, and weighed 88 grains. Its nucleus appears to have been formed in the lacrimal sac, whence it descended.

Assistant-Surgeon K. K. Mitter⁵ extracted, under chloroform, a small oval calculus, with a hour-glass contraction in the middle, which was firmly adherent to the mucous membrane of the ala of the nose of a native girl of five.

¹ *C. M. T.*, vol. iv. p. 31.

² *I. M. G.* for January 1885.

³ *C. M. T.*, vol. ii. p. 306.

⁴ *I. M. G.*, vol. viii. p. 43.

⁵ *Ibid.* p. 156.

S. Gozdar treated ¹ a native man, *æt.* 25, who had suffered for years from obstinate catarrh, confined to the left nostril, with severe neuralgia-like pain of that side of the face, attributed to a blow with a stone which broke into pieces. A stone, doubtless a rhinolith, one-and-a-half inches in length and nearly three in girth at its thickest part, was removed.

It is probable that *Foreign Bodies* usually form the nuclei of these concretions when they occur in children. I had considerable difficulty in clearing the nostrils of a boy of four, who, if still living, is a baronet, from masses of lime from the terraced floor, which he had passed the morning in cramming in.

I met with a healthy and eminent Scotsman in India who considered that, although he believed that he had full powers of taste, the sense of smell was absent from birth.²

Thirty-seven years ago, an Indian civilian informed me that, a year or two previously, he had sustained a dangerous injury to the head—probably fracture of the base—which left him without sense of smell. I believe that he is now living.

DISEASES OF THE EAR.

The Bengal Census return of 1881³ gives 12·32 per 10,000 of *Deaf Mutism*, against 5·07 in England. It has been recently stated that there are upwards of 500 of these unfortunates in Bombay in a population of 773,196.

A poor woman, of the Koonjur class, applied to Dr. R. F. Hutchinson saying that she was outcasted because the largely perforated lobe of her right ear had been accidentally torn through. Two snips with scissors, removing the cicatrised ends, and one suture did the needful for her and restored her to her tribe.

Pendulous Tumour.—Dr. A. Campbell and Mr. M. J. Bramley have described ⁴ a ‘Pendulous Tumour of the External Ear.’ Both ears are usually affected. The tumours arise from any portion of the external ear. The growth is so rapid that a tumour two months old can seldom pass unobserved. These are liable, when growing from the helices, to draw down and double over the ears so as greatly to obstruct hearing. They may be so large as to reach the shoulder.

¹ *I. M. G.*, 1882, p. 341.

² Harriet Martineau did not possess the senses of taste and smell. She said, ‘I tasted a leg of mutton and it was delicious.’ The gift was withdrawn as suddenly as it came. The sense of smell was denied to Wordsworth. In his case, too, it was vouchsafed to him only on one occasion. ‘He once smelt a beanfield and thought it heaven.’—James Payn’s *Literary Recollections*.

³ Cited in the *I. M. G.*

⁴ *C. M. T.*, vol. vi. p. 488, and vol. vii. part 1, p. 71.

A pair, removed from a woman, weighed two pounds. Men, women, and children are alike subject.

Dr. Campbell described this as a very frequent disease in the Valley of Nipal, often occurring in the subjects of goitre. His inquiries, as far as he was able to carry them, led Mr. Bramley to believe that it only occurred in Neel Kheut, a small village which, with its environs, may occupy a square mile. In the first volume of the 'Calcutta Medical and Physical Transactions,' Dr. John Adam described and figured a pendulous tumour of the integument, surrounding the outer ear, which rode upon the shoulder, and descended behind and before, reaching, at last, the plane of the nipple. It was clearly *molluscum*. I have alluded, in the chapter on *Leprosy*, to the tuberculated hypertrophy of the lobe of the ear which is so almost invariably a characteristic of that disease.

Ear-Ache, from diffused or circumscribed inflammation of the external meatus, is not infrequent in India.

A case, published in April 1884, makes me aware that laudanum with oil has not yet gone out of use as a soothing application. In cases of ear-ache, &c., this 'venomous distilment' acts like liquid fire upon the sensitive mucous membrane. I always used pure opium rubbed up with olive oil. The application of two or three leeches, precisely in the cleft between the mastoid process and the ear, and the free use of steaming, by means of a jug of hot water and a towel, will, I believe, nearly always render incision needless.

When I was civil surgeon of Howrah, opposite to Calcutta, in 1853, *Inflammation of the External Meatus* giving rise to *Chronic Otorrhœa*, was a widely spread endemic among natives, thousands of cases occurring along the right bank of the river. I did not see any case in a European, nor could I learn that the disease was prevalent on the Calcutta bank. In the Mutiny, a young brother officer contracted the disease when sleeping on the ground at Barrackpore (on that side of the river) and lost hearing in that ear.

Dr. W. J. Moore¹ speaks of diseases of the ear as being remarkably common in Joudpoor. Children especially suffer from these complaints, and deafness of one or both ears is very frequent. The affection generally commences by inflammation of the external auditory canal, which, being neglected, leads to extension of the disease inwards, perforation of the membrana tympani and destruction of the internal apparatus. It appears to originate from irritation produced by the fine particles of sand which, during the hot weather, load the atmosphere and are deposited in the aural passages. Instead of frequently washing and cleansing the parts, inserting wool is a common practice. This tends to extend rather than allay irritation. In the

¹ I. A., No. xx. p. 525.

early stages of the affection, frequent washing and gentle syringing will effect a cure.

Not long ago, an acquaintance of my own, in London, excited very troublesome chronic irritation in the meatus by picking his ear with a pin. The following note, although old,¹ conveys a useful hint. Mr. McGregor, of H.M.'s 39th Regiment, observed: 'A custom prevails among the soldiers, and in fact among the Europeans in general, to which is attributed, in part, the occurrence of otitis. Instead of everyone performing his own "tonsonial matutinal operations," the native barber caste are usually called into play, by whom shaving is daily performed. This being finished, the ears are next cleaned, and the cerumen removed, sometimes too roughly. Either from this cause, or from cold affecting the cleaned ear, the disease in question frequently occurs. All did well except one case, in which the inflammation terminated in purulent discharge, and extended to the osseous case of the ear, and ultimately to the membranes of the base of the brain.'

Mr. G. C. Hall speaks² of *Bather's Ear-ache* as a very common affection in India, wherever there is a swimming-bath. He attributes it to diving, the pressure of the water causing small foreign bodies to be carried into the meatus, causing inflammation. It occurs especially in those who dive much. The symptoms given are those of catarrh of the meatus, sometimes going on to abscess. Mr. Hall uses opium and glycerine and 15 grains of alum to an ounce of glycerine warmed and poured into the ear.

The water of a bathing-place near Athens is said to cause ear-ache.

Maggots in the Ear.—At Chittagong, I attended an officer's daughter, aged 18, who was a paralysed and bed-ridden idiot. The meatus of one ear was suppurating and filled with large maggots. I used various lotions [I learnt, at Guy's, not to syringe the ear, but to place the head flat on a low pillow and to fill the meatus and concha with warm water or medicated fluid, which was allowed to remain there as long as might be needful] which did not appear to trouble the parasites at all. I, therefore, removed them with dissecting forceps, all of them clinging on and resisting with great tenacity. In doing this, I found that the bony canal was quite bare. The meatus was lightly plugged and dressed regularly. The surfaces granulated, and healing went on to such an extent that the cicatrised meatus remained as a round hole two-thirds smaller than was natural. At the same time in the following year, I was called to her again and found her moribund; the other meatus was filled with maggots.

In the article cited above, Dr. Moore, in describing *Peenash*, as it occurs in Marwar, says: 'Similarly, I have occasionally met with maggots in the ear, after otitis and perforation of the tympanum.'

¹ *Madras Quart. Med. Jour.*, vol. ii. p. 375.

² *I. M. G.*, December 1876, p. 315.

Polypus.—During the four years ending 1883, the returns give admissions as follow: European soldiers, 27; native soldiers, 2; prisoners, 4. The disease did not occur among the European women and children.

Foreign Bodies.—At Howrah, an East Indian boy, of 4, was brought to me with a custard apple seed far back in the meatus, which was a good deal inflamed and swollen. This spindle-shaped seed is as hard, polished, and slippery as a piece of smooth carnelian of the same form would be. It took me nearly an hour to hook it out.

Assistant-Apothecary Adie reported¹ the case of a native male child which had a pea in the meatus for eighty hours, the passage being much swollen. He broke it in two with a sharp probe and so removed it. The handiest instrument for this purpose is a lady's black wire hair-pin bent into the form of a scoop.

The fact that I only observed *Disease of the Mastoid Cells* once, in a Mohammedan woman, is probably due to the immunity from struma of the natives of Lower Bengal. In the four years cited above, the returns give 5 cases of this condition in European soldiers: 2 in European children, 2 in native soldiers, and 1 in a prisoner.

DISEASES OF THE CIRCULATORY SYSTEM.

As I have described elsewhere² most of the principal diseases of the heart and bloodvessels, I shall restrict myself here to giving a few notes of cases occurring in India.

¹ *I. M. G.*, December 1875.

² 'A Collection of Facts Illustrative of the Diseases of the Pulmonary Artery,' *London Medical Gazette*, 1845 to 1851; and privately printed, London, 1851.

'On the Diseases of the Orifice and Valves of the Aorta,' *Guy's Hospital Reports*, vol. vii. p. 443.

'On the Structure and Diseases of the Coronary Arteries of the Heart,' do.

'On Acute Aortitis,' do., vol. vi. for 1841, p. 304.

'On the Structure of the Subserous Membrane of the Aorta,' do., vol. v. p. 40.

'On Permanence of the Ductus Arteriosus and Contraction of the Thoracic Aorta, and on the Means by which the Duct becomes Naturally Closed,' *London Medical Gazette* for May 30, 1845.

'On the Effects of Ligature of the Carotid Arteries upon the Cerebral Circulation,' *Ibid.* for 1845, vol. xxxvi.

On the same subject, *I. A.*, No. ii. p. 706.

'General and Aneurysmal Dilatations of the Aorta, especially with Reference to their Pathology and Treatment,' *London Medical Gazette* for August 1845, pp. 725 and 769.

A Practical Treatise on the Management of Diseases of the Heart, and of Aortic Aneurysm, with Especial Reference to the Treatment of those Diseases in India, Calcutta, 1851.

'The Structural Anatomy of the Veins,' *London Medical Gazette* for August 8, 1845.

Congenital Malformations of the Heart are not frequently seen in India, in all probability only because native children thus affected are not brought to us, and usually die early. Prep. C 50 in the Grant Medical College collection represents *Contraction of the Orifice of the Pulmonary Artery*, probably congenital. The heart weighed $7\frac{1}{2}$ ounces. The orifice seemed contracted by adhesion of the free edges of the semi-lunar valves. The trunk of the pulmonary artery was of diminished capacity. The right auricle was alone dilated. The patient died at the age of 15, having suffered pain and dyspnoea from childhood.

I was more than once consulted in the case of a Jewish girl in whom signs and symptoms of stenosis of the pulmonary ostium were marked. She died, after menstruation had been established, from arterial gangrene of both feet. I also observed evidences of the same defect in two officers' sons who did not survive puberty. These boys were not at all cyanosed. I found only two properly formed aortic sigmoids in a European infant which died of atelectasis pulmonum. Dr. William Mortimer published¹ the case of an Indo-Briton, aged 23, an ordnance artificer of healthy appearance, in whom the orifice of a two-valved aorta had, by thickening of the abnormal structures, become obstructive. I long ago adduced evidence of the law that the structures of congenitally malformed valves, &c., are especially prone to suffer further deterioration by endocarditis.

Prep. 709 in the College Museum displays a similar want of one of the pulmonary cusps.

In Prep. 710, from a native man, there are four pulmonary cusps. The supernumerary valve is scarcely half as large as the others.

Prep. 58, Ser. 17, in Mr. Sibthorpe's Madras catalogue is a pulmonary artery with four semi-lunar cusps from a man—race not specified.

There are other valuable examples of cardiac malformation described in the Calcutta catalogue; 706 is the heart of a new-born infant in which there is remarkable stenosis of the pulmonary orifice, with patency of the ventricular septum, ductus arteriosus, and foramen ovale; 508 is the heart of an infant, five months old, with patent arterial duct and foramen ovale. I had, preserved in the museum, a very fine dry specimen of transposition of the heart and other thoracic contents from a young native woman who died in my ward. The abnormal position of the heart was clearly visible during life.

Mr. Heming recorded² the case of a native male child, aged two years and eight months, which was markedly cyanotic. It is evident that the report of the condition of the heart is imperfect, but the

¹ *Madras Quarterly Medical Journal*, vol. ii. p. 65.

² *I. M. J.*, vol. vii., N. S., p. 345.

ventricular septum was patent, the foramen ovale closed and the arterial duct open.

Dr. A. Garden has described¹ the appearances found in a well-formed, plump, and healthy-looking native female infant stated to have been still-born. The left half of the diaphragm was deficient. The small intestines occupied the upper two-thirds of the thorax, a portion of the colon reached almost up to the clavicle. The stomach lay across the lower part of the thorax, behind it was the spleen, part of the liver also lay in the thorax. The heart lay on the right side; the aorta arched backwards from right to left. The right lung was moderately developed. The undeveloped left lung, although seemingly normal in structure, was about the size of half a walnut. No. XV. of the 'Indian Annals of Medical Science' contains a valuable paper on 'Malposition or Displacement of the Heart,' by Dr. W. Curran.

When at Howrah, about the year 1853, happening to mention to Mr. Burgess that, although I had seen nearly every form of congenital defect, I had never met with a case of *Branchial Fistula*, he told me that there was then a large Christian family in Calcutta, most of whom displayed this peculiarity. Many years afterwards, I saw a European with branchial fistula. There was a small opening half-way down the neck, in front of the sterno-mastoid muscle, into which a probe passed deeply about two inches. At first sight, it looked very like a sinus, but it had a distinct mucous lining.

Dr. T. A. Wise has related and figured² the case of a strong middle-aged native who died suddenly while walking. Below the origin of the left subclavian artery and the completely impervious arterial duct, the aorta was absolutely closed as if a ligature had been placed tightly round it. Immediately below this point, the aorta appeared of its natural size, and into it 'several large arteries poured in their blood.' A dissecting aneurysm had been caused by a rupture of the walls of the aorta a little above the semilunar valves. This had given way, causing fatal hæmorrhage into the pleural cavity.

A very remarkable abnormality in a female infant, one of twins—still-born at about the eighth month—is noticed³ by Mr. G. McReddie. It appears that the pulmonary artery did not give off any branches to the lungs, but was continuous with the aorta by a widely open arterial duct. The aorta gave off a trunk which, doubtless dividing into two, proceeded to the lungs and acted as a substitute for the normal branches of the pulmonary artery.

I have always looked upon the typical 'button-hole' mitral as a congenital malformation. I met with a case in which, with this condition, the *tricuspid orifice*, viewed from the right auricle, was also nearly in the button-hole condition.

¹ *I. M. G.*, vol. v. p. 55.

² *M. P. T.*, vol. viii. part ii. p. 385.

³ *I. M. G.*, January 1866.

Cardiac Debility.

There exist in India all those conditions—with the exception of senility in Europeans—which lead in other countries to weakening of cardiac muscular tone—fevers and other exhausting diseases, inordinate tobacco-smoking, scorbutus, anæmia, &c., &c. I have spoken¹ of *Cardiac Asthenia* as a result of paludal poisoning. As, happily, most of those who suffer from this affection in Lower Bengal recover on taking change to Europe, I have not been able to judge precisely whether they are the subjects of weakness of cardiac muscle or of a neurosis; our knowledge of another Indian neurosis, malarial asthma, leads me to incline to the latter view.

Mr. Charles Macnamara has recorded, in Part V. of the 'Indian Annals of Medical Science,' a series of important observations upon the tendency which inactivity and over-feeding have to produce fatty changes in the heart and other organs of young soldiers in India. His remarks are equally applicable to all classes of men employed in that country. Many deaths of Anglo-Indians, in and out of India, are indirectly due to cardiac debility, predisposing to rapid asphyxia, in various states of disease. The cases of several persons, whose constitutional states were known to me, recur to me as I write. A, an anæmiated man, died at Rome of pneumonia; B, an immoderate smoker, at Paris, of enteric fever; C, a 'liver-grown' individual, as soon as he reached Europe, of bronchitis; D, a subject of malarial asthma, on return to London, of what would, probably, in a sounder constitution, have been merely catarrh; E, a man known to have a weak heart, soon after entering the rarified and cold atmosphere of a hill-sanitarium. Had the muscular tone of their hearts been duly conserved, these individuals would probably have been living now.

Rheumatic Fever with Cardiac Complication (see p. 322) occurs distinctly, but very rarely, in Lower Bengal. I believe that it is more frequent northward. On an average, I did not treat more than one case annually in my sixty beds. I can only recall one fatal case in my own practice, and another, a European officer, which I saw in consultation. My patient, a native lad, got pericarditis and then pleurisy, and so died. The officer had pericarditis, but I think that there was no *post-mortem* examination.

In 1839, Dr. Shanks, of H.M.'s 55th Regiment, observed that disease of the heart does not appear to be so usual a concomitant of articular rheumatism in India as in Europe. Commenting upon this, the editor of the 'Indian Journal of Medical and Physical Science'² remarked that he could call to recollection some well-marked cases in India.

¹ *Vide antea*, p. 241.

² Vol. iv., N. S., p. 561.

Writing in 1835 from Bangalore, Mr. Hamilton of H.M.'s 39th Regiment, noticed that, although rheumatism had been rather frequent and severe, requiring active treatment, there was no distinct pericardial or heart complication.¹

Reporting from Bellary (Madras Presidency), in 1840, Dr. Davis, of H.M.'s 39th Regiment, wrote: 'Rheumatism, in an uncomplicated form, seldom proves fatal in India, and is unaccompanied by the cardiac affections so frequently met with in Europe.'² It is stated, in a report from Arcot,³ that, in one instance only of 241 cases of acute rheumatism, 'metastasis' took place. It is added, 'this character of rheumatism, so frequent in Europe, is rarely observed in India; and, amongst natives, it may be said never to occur; in Europeans, likewise, it is seldom seen except in young men recently arrived, who have generally contracted the disease on board ship. In the case above alluded to, the 'metastasis' did not affect any of the internal organs, but the disease shifted from one joint to another. Commenting upon the death of a European artilleryman of Hyderabad, Deccan, Dr. George Smith⁴ wrote that the connection between acute rheumatic disease and pericardial inflammation is too often overlooked in India, from the idea, correct in the main, that rheumatism in the European soldier is less frequently complicated with heart disease there than in England. The very great frequency of this serious complication in Britain, and the experience of some medical men in India, should, he urged, put us upon our guard in all cases of acute fibrous rheumatism occurring, especially for the first time, in young and healthy men, and should stimulate us to watch carefully the earliest symptoms of the approach of the disease. Morehead remarked that '*acute* articular rheumatism is not so common in India as in colder climates, yet it is by no means rare'; and a complicating pericarditis or endocarditis is, he believed, as frequent an occurrence in the one country as in the other. He added, 'Of no rule of practice am I more thoroughly convinced, that it is as incumbent on the practitioner in India as in Europe carefully to watch and search for the physical signs of pericarditis and endocarditis in every case of acute rheumatism. If this rule be neglected, the co-existence of these diseases in India will necessarily continue to be considered an unusual event.' Dr. C. Alexander Gordon, then of the 10th Regiment, commented upon the above observations,⁵ and Dr. Morehead replied,⁶ Dr. Gordon being of opinion that endocarditis and pericarditis very rarely, if ever, occur in India during the attack of acute rheumatism; that Queen's medical officers were most careful to watch for the physical signs of these affections, and that the proportion of soldiers, invalided

¹ *Ibid.* vol. v. p. 602.

³ In the *M. T. R.*, p. 37.

³ *I. A.*, No. 11, p. 7.

² *M. Q. J.*, vol. iv. p. 6.

⁴ *I. A.*, No. 8, p. 635.

⁶ *Op. cit.* p. 562.

in consequence of disease of the heart in India, is not a tithe so large as in the United Kingdom. Dr. Morehead held that this last observation seems to prove the comparative rarity of acute rheumatism in India, rather than the infrequency of pericarditis as a complication. His opportunities of judging of the frequency of cardiac disease in European regimental hospitals were not few, and many cases of the complication of cardiac disease with acute rheumatism had come under his observation. In his XXVth chapter, Morehead records the clinical history of 28 cases of structural disease of the heart, and 3 of aneurysm of the aorta *in natives of India*. In 12 of the 28 cases, it appeared that 'rheumatism' had been stated to exist.

The experience of a working life-time has taught me that acute rheumatism is rare in Lower Bengal; in the majority of the very few cases which came under my notice, there was cardiac complication.

No one can object more strongly than I do to mere guess-work in diagnosis. Still, it may, I think, be very generally predicted that heart-symptoms—placing aside those of aortic aneurysm—in one long resident in India, are probably not due to serious organic mischief. The history of acute rheumatism with cardiac complication is usually wanting in these cases.

Acute rheumatism with its cardiac complications appears to be rather frequent among old Indians retired in England. During the last ten years, I have heard of six cases, in three of which the heart appears to have been affected, in my own circle of acquaintance.

The thirteen preparations, marked Nos. 569 to 581, in the Medical College Museum illustrate *Pericarditis* and its effects. Five of these were sent from distant stations. In No. 569, the pericarditis was attributed to violence. It is probable that some of these specimens were from European sailors.¹

Preparations 608, 609, and 610 well represent displacement of the heart to the right by left empyema. The first and second are from natives, the third is from a European.

Before I went to India, I entered into a rather painful discussion

¹ I have always valued the Museum Catalogue of the Calcutta Medical College exceedingly. It is the work of two of India's ablest pathologists. In producing it in 1865, Dr. Joseph Ewart, now of Brighton, may be said to have re-edited that portion of Allan Webb's admirable *Pathologia Indica* which describes the preparations still in existence. All more recent preparations are described by Dr. Ewart. Since I have worked at the Catalogue, I have noticed a defect for which its authors are in no way to blame. In many cases it does not appear (1) whether the specimens are from Europeans or natives, and (2) whether the lesion represented certainly or probably originated in or (as in the case of European soldiers, sailors, &c.) out of India. This being a great fault in the description of an invaluable collection which must now number more than two thousand typical specimens, I trust that, as new preparations are added, my successors will provide that these points shall be made as clear as possible.

in maintenance of a claim to the observation that, although hypertrophy and dilatation of the heart are seen in the majority of cases of old pericardial adhesion, the natural tendency of this state of the pericardium is to produce atrophy of the heart where the exit of blood from that organ is not impeded by concurrent valvular stenosis or obstruction to the pulmonary or systemic circulation. Preparation 13 markedly illustrates this pathological law. It represents atrophy of the heart consequent upon old-standing adhesion of the pericardial surfaces. The agglutinating material 'consists of three alternating layers of white and brown structure. It is cartilaginous in consistency, and, inclusive of the thickened pericardium, measures *about half an inch* in thickness, whilst the subjacent wall of the left ventricle varies only from the *sixteenth to the eighth of an inch* in thickness. The carneæ columnæ, chordæ tendineæ, and curtains of the mitral valve are also much attenuated. The aortic valves are healthy.' The wall of the right auricle is only the sixteenth of an inch thick, whilst 'the altered and thickened pericardium is fully thrice as thick.'

The only good that came out of this unpleasant affair was a nobly candid expression by Dr. Thomas Addison. While the contest was still recollected, a paper on pericarditis was read at a meeting of the Physical Society of Guy's Hospital. The dangerous point was avoided in the discussion until my opponent stated his case, just as we were about to separate. Dr. Addison rose and proposed that the discussion should be continued. He said that he had, of course, often noticed, with others, that cardiac hypertrophy and dilatation were attendant upon old pericardial adhesion, but he had seen cases in which the heart was small and atrophied. He had never been able to reconcile these observations until Dr. Chevers gave the explanation.

I frequently met with extensive traces of old *Carditis* and *Endocarditis*, not obstructive, in examining the bodies of native hospital patients.

Cases of *Liver Abscess opening into the Pericardium* will be found in the section on 'Hepatic Disease.'

A very remarkable case of *Rupture of the Pericardium* has been reported¹ by Dr. Lewtas. A Calcutta cooly fell into the hold of a ship and died in two hours after admission. There was a rent, two inches long, on the right side of the pericardium, its edges were blood-infiltrated. There was another similar but smaller rent nearly corresponding with the apex of the heart. There were six ounces of blood in the right pleura, doubtless from the rent pericardium.

This case is very important as showing that an extremely severe blow to the heart does not necessarily cause immediate death by nervous shock. The following case appears to indicate that the rents in the pericardium were caused by squeezing the heart.

¹ I. M. G. for November 1876.

In a cooly whose chest was crushed between the buffers of two railway carriages, Mr. John O'Neill found that the heart had been squeezed off through a hole at the apex of the pericardium, and lay quite detached, resting on the diaphragm at the extreme left of the thoracic cavity, beneath the left lung.¹

Prep. A 158 in the collection of the Grant Medical College represents a very unusual injury to the pericardium. Fracture of the sternum extending obliquely across the bone from the fourth left sternocostal articulation to the fifth on the right side. The patient was admitted with severe shock, dyspnoea, and a slight external wound, the result of a fall upon a newly-metalled road. Death on the eighth day. The anterior surface of the pericardium had a laceration about three quarters of an inch long. The pericardial branch of the internal mammary artery had been wounded, and thirteen ounces of blood had been effused into the pericardium.

At page 409 *et seq.* of my 'Medical Jurisprudence for India,' I have cited several Indian cases of *Spontaneous and Traumatic Rupture of the Heart*. There are two, by Drs. Hutchinson and Simpson, in which a blow with a club or *lattee* (long male bamboo) ruptured the heart. A similar case has since been reported.

In the Calcutta Medical College Museum, Preparation 604 is one of spontaneous rupture of the *right ventricle* in a native, a very curious, but, to me, obscure case.

Preparation 605 represents rupture of the *right ventricle* of a native's fatty heart, presented by Dr. Mountjoy. My friend, Dr. Theodore Duka, has described² the case of a Mohammedan, *æt.* about 65, who was reported to have had a fall during a scuffle, after which he suddenly expired. There was a *rupture of the pericardium* over the apex of the heart, about three lines long and running horizontally. Its edges were ecchymosed to the extent of about two lines. There was a considerable quantity of uncoagulated blood in the left pleural cavity; the pericardium was also distended with blood. There were two rents in the *right ventricle*, near the septum; one of these opened the coronary vein. The whole organ presented extensive fatty degeneration.

Preparation No. 606 in the College Museum is one of fracture of sternum and ribs, with *laceration of the pericardium* and rupture of the *left ventricle* from a native who died within a quarter of an hour after having been run over by the wheel of a buggy.³

Mr. McReddie examined the body of an apparently able-bodied healthy native man who was found dead on his threshing floor. There were a deep ecchymosis over the third and fourth left costal cartilages, rupture of the spleen (apparently) and a rent in the *left ventricle*. These injuries were probably incurred in a struggle with thieves.⁴

¹ *I. M. G.* for 1878, p. 44.

² *I. A.*, No. xv. p. 257.

³ *C. M. T.*, vol. vi. p. 476.

⁴ *I. M. G.*, vol. iv. p. 141.

Mention is made in my 'Medical Jurisprudence' of the case of Israel Hinton, in whose heart a musket bullet occupies the apical part of the cavity of the left ventricle. This case has been narrated by Sir Joseph Fayrer.¹ At the storming of the Great Pagoda at Rangoon on April 14, 1852, this poor fellow was struck a little above the anterior fold of the left axilla. He died, worn out by pulmonary disease, on June 24. The preparation, numbered 607, is one of the most valuable in our Medical College Museum. The ball is described in the catalogue as being 'almost completely embedded in the muscular structure of the left ventricle at its apex.' It appeared to me rather that the bullet, having dropped into the cavity of the ventricle, had, by its weight, formed a sac for itself. There was a canal in the middle of the consolidated left lung in which was a small portion of the jacket cloth. 'This canal, passing forwards and inwards, stopped short close to the union of the pulmonary veins.' Unfortunately, the pulmonary veins, through one of which, in all probability, the ball found entrance to the heart, are not preserved. Dr. Burton Browne and I most carefully examined the preparation, and could not discover the slightest trace of a route by which the bullet could have entered the heart.

Dr. Curran related to me the case of a native man, at Peshawur, who, while sleeping, was approached by a fanatic and shot in the heart with seven slugs. He lived long enough to identify his murderer.

Mr. Galt reported the case of a sepoy who died suddenly while under treatment for fever in hospital. His age is not mentioned, but he could not have been old. The description is not clear, but I think that there must have been an *Aneurysm*, which ruptured at the *apex of the left ventricle*.

Sir W. O'Shaughnessy Brooke brought before the Medical and Physical Society of Calcutta, in 1836, *an aneurysm*, about the size of a walnut, projecting from the *apex of the left ventricle*. The coats of the sac were thin, and it was evidently formed by the endocardium protruding through the muscular walls. The subject of this lesion, a native, died suddenly in the street, the sac having burst and caused hæmorrhage into the pericardium.²

The specimen numbered 599 in the Calcutta Medical College collection is a 'heart showing two *aneurysms* in the walls of the left ventricle near the apex.' Each of these is about the size of a walnut, and communicates by a comparatively narrow opening with the cavity of the ventricle. The margins of these orifices are smooth, and lined by an opaque-looking membranous structure which is continuous throughout the interior of the aneurysms, both of which have led to the entire absorption of the muscular structure, being bounded on their external aspect, which projects half an inch above the level of the cardiac sur-

¹ *Lancet*, May 10, 1879, p. 658.

² *I. M. J.*, vol. vi., N. S., p. 814.

face, by thickened and adherent pericardium. In some parts this shield or barrier is a quarter of an inch in thickness; whilst, in others, it does not exceed the tenth of an inch. Each of the sacs was filled with fibrine in process of softening and degeneration, and a portion of coagulum is now seen in one of them. The patient was admitted into the Calcutta Native Hospital in a dying state.

There are three specimens of aneurysm of the heart in the Madras collection: ¹ (1) Aneurysmal dilatation of the apex of the left ventricle about the size of a duck's egg. The orifice of communication between the sac and the ventricle measures three quarters of an inch in diameter. (2) Heart containing a bony cyst situated in the wall of the left ventricle. The patient was admitted dying with symptoms of uræmic poisoning. The cyst is about the size of a hen's egg and communicates with the cavity of the ventricle by an oval opening which has sharp edges. Its interior is filled with clot. 'It is probable that a cardiac aneurysm originally existed, which has subsequently become ossified.' The liver and kidneys were diseased.

(3) Preparation 144, Appendix, p. 140, shows a cardiac aneurysm in the left ventricle which burst into the pericardium.

Mr. John Adam published ² the case of an intemperate military invalid, *æt.* 52, a native of Switzerland, who died suddenly in India. It was found that a large quantity of fluid blood had been effused into the right pleura. The pulmonary valves 'were in a state of ossification. The *pulmonary artery* was extremely thin and enlarged to nearly four times its natural size.' It contained 'an organised polypus extending from the semilunar valves about four inches along the artery, of which the circumference was not less than seven inches.' 'The *rupture* had taken place in the middle' (doubtless half-way up the pulmonary trunk). The aorta and its branches 'were completely ossified.'

A case of *Gummatous Tumour* of the heart occurred to my friend, Dr. Francis, while officiating for me at the Medical College Hospital in 1872.

There is a very slight and imperfect notice of an *Abscess of the Heart* reported by Mr. Knight to the Medical and Physical Society of Calcutta. During life, the patient showed no sign of cardiac affection or severe disease of any kind.³ No. 602, in the Medical College Museum, is described as '*an abscess of the septum ventriculorum of the heart.*' It was presented by Professor Wilson.

After a life-long search for varieties of heart disease, I know little or nothing of abscess occurring in the cardiac walls except as a pyæmic deposit. I suspect that, in some cases, those putty-coloured fibrinous concretions, described by Dr. Henry Marshall Hughes in vol. iv.,

¹ Vide Sibthorpe's Catalogue, Preps. 22 and 40, Series 5.

² *C. M. T.*, vol. ii. p. 114.

³ *I. M. J.*, vol. iii., N. S., p. 767.

p. 146, of Guy's Reports, have been mistaken for abscesses. They are hollowed, and are filled with a thick pasty fluid which is mainly broken-down fibrine, but which has, to the naked eye, a puriform appearance. They evidently have a degree of organisation, as I showed in the 'Medical Gazette' for August 1845, p. 774. In a case of sea scurvy which proved fatal in my ward, I found several of these concretions—one of rather large size occupied the right auricular appendix.

Not to speak of cases of mitral and aortic stenosis arising from ordinary endocarditis, I saw, in India, more than one example of *Congenital Narrowing of the Mitral Orifice*. I presented to the collection Preparation 623: 'button-hole constriction of the mitral orifice.' The opening is a mere slit, from (congenital?) thickening, puckering and contraction of the curtains of the mitral valve. From a native female, 25 years of age, who died suddenly, Preparation 621, is one of mitral stenosis from 'calcareous degeneration.' The slit is so narrow as to render the introduction of the little finger difficult. It was from a native of Bengal who died with general anasarca; presented by Professor Mouat. Professor E. Goodeve presented Preparation 622: 'button-hole mitral constriction. The orifice will scarcely admit more than a common pencil.'

I met with several cases, in Calcutta, in which there was clear evidence during life of *Regurgitant Disease of the Aortic Valves*, in a native merchant, in a stoutly-built middle-aged European woman, in a very powerful European. The last-named was employed in supervising the construction of the great drainage culvert which runs down Dhurumtolla. In this work, bricks were not laid down singly, but several were placed with mortar in a wooden frame and allowed to consolidate—thus masses weighing nearly a hundred-weight were formed. My poor patient was in the habit of taking these ponderous bricks in his hands and building them in. On one occasion, he felt something give way in the præcordial region and came to hospital, where he died. The European woman's case was one of rather unusual complexity. I sent the diseased structures to our museum (Preparation 631). The aortic valves are somewhat thickened, rigid, shallow, and contracted, and one curtain is quite incompetent to prevent the reflex of blood during the diastole. The upper margin of another shows a tendency to retroversion. There is, consequently, a dilated and hypertrophied left ventricle. The curtains and muscular columns of the mitral are also thickened, but efficient. The ascending aorta is slightly dilated, and this, as well as the arch and descending portion, are partly atheromatous. About an inch above the faultiest cusps, there is a small aneurysmal pouch, about the size of a hazel nut, which is advancing towards the descending cava at its termination in the auricle. The internal and middle coats, at the apex of the tumour, have given way, and there is only the attenuated cellular coat prevent-

ing the extravasation of blood. The congestion of the cellular coat of the cava, just where rupture was impending, indicates the commencement of the process of exudation of organisable material wherewith nature might have delayed the rupture of the aneurysmal sac. There was a double bruit above the aortic valves. It would have been doubtful whether this depended upon aneurysm of the ascending arch, or upon not excessive regurgitant disease of the aortic valves. The moderate water-hammer character of the pulse decided this point. While in India, I more than once found a double murmur in the ascending aorta without regurgitant diastole. Here there was probably either aneurysm of the ascending aorta or great roughening of its walls, the valves being efficient.

Diseases of the Coronary Arteries.—Preparation 632 displays calcareous degeneration of the coronary arteries in an old enfeebled Bengalee. There was also general atheromatous disease of the arteries. The coronary arteries are 'quite ossified like quills.' The heart is atrophied.

Unfortunately, the race to which the subject of Preparation 633 belonged is not stated. It is a very remarkable specimen of *Aneurysm of one of the Coronary Arteries*. There is atheromatous deposit in and thickening of the semilunar valves, and in the lining membrane of the sinuses of Valsalva. The orifice of the coronary artery leading to the aneurysm is large enough to admit a goose-quill. About half an inch down, is the sac of the aneurysm, which is about as large as a small walnut. Its walls consist of the thickened visceral pericardium, the altered cellular tunic of the artery, and a more or less interrupted lamina of organised lymph. There is a good deal of thickening and opacity of the pericardium for about half an inch around the circumference of the aneurysmal sac.

Mr. Charles Macnamara¹ saw two cases of European soldiers, the subjects of general fatty degeneration, in whom *the aorta had ruptured*, in one case *within*, and in the other just *outside* the pericardium. 'There was no dilatation of any part of the vessel, but, owing to an excessive deposit of fat from the vasa vasorum, the walls of the vessels had become infiltrated with fat, interfering with their due nourishment and rendering them incapable of resisting the force of the blood, and they had burst at the point where they had become much degenerated.'

I saw a similar case in the dissecting-room at Guy's in the extremely diseased and much dilated, but not aneurysmal, aorta of an elderly man. The vessel was rent within the pericardium.

Preparation 66, Series 5, in the Madras Catalogue shows rupture of the fore part of the aorta at its commencement. The opening easily admits a finger. The coats of the artery in its vicinity are thinner

¹ *I. A.*, No. viii. p. 182.

than elsewhere. The pericardium contained about twenty-four ounces of fluid and a clot of blood.

In my subsequent practice, I adhered to the following rules, which I recommended in 1852¹ as the leading indications of *Treatment* in cardiac disease.

1. To diminish, if possible, the valvular or other immediate causes of obstruction.

2. To endeavour to remove all causes of impediment to the circulation existing in the lungs, abdominal organs, and capillary system generally.

3. To lessen vascular distension by reducing the bulk of the circulating fluid, without impoverishing the system.

4. To sustain or restore the power of the heart, and to reduce the capacity of its dilated cavities.

5. To equalise the circulation, and to maintain free vascular action on the surface by regulating the temperature, clothing, &c., and to procure, as far as possible, rest and tranquillity of body and mind.

See remarks on *Cardiac Anæmia*, p. 253.

Aneurysm.

During the past forty years, I have been allowed full opportunity of regretting that my medical brethren should neither have controverted nor adopted the observations which I published in 1845, on the causation of those 'true' aneurysms which are dependent upon dilatation of the arterial walls.

They were not mere theoretical speculations, but were the outcome of much attentive study of arterial disease in the dead-house and in museums. All my subsequent observation has confirmed their general accuracy, and I venture to believe that they still deserve attention.

I think that we, the young morbid anatomists of 1845,² were not uninstructed in the subject of '*Arterial Tension*,' vide Dr. Goodhart's admirable Bradshawe Lecture for the year 1885. Our writings show that, in the system of the gin drunkard, we had learned to perceive the existence of chronic blood impurity maintained by a constant supply of turpentine, juniper, white-vitriol, and alcohol. Hence the over-taxed kidneys and liver became diseased. Impediment to the circulation through their capillaries gave rise to undue tension resulting in dilatation and hypertrophy in the arteries and left heart—we assigned far more importance to the dilatation than to the hypertrophy, viewing the latter rather in its compensatory than in its morbid aspect.

¹ *Management of the Diseases of the Heart, &c.*, p. 13.

² 'General and Aneurysmal Dilatation of the Aorta; especially with reference to their Pathology and Treatment,' *London Medical Gazette* for August 1845, pp. 725 and 769.

Imperfect elimination of urea and bile elements produced a tendency to endocarditis and degeneration of arterial tissue. We had made a special study of the various conditions which lead to morbid tension in the pulmonary artery. The dependence of permanent dilatation of that vessel and of the right heart and venous system, leading to dropsy, upon over-work, intemperance, chronic bronchitis, emphysema, mitral disease, &c., was a matter of daily observation.

Aneurysm of the Ascending Aorta.—Preps. 636 and 638 represent aneurysms of the ascending arch in natives.

An apparently healthy captain, having taken his usual morning walk, returned to his quarters at the Military Club, Calcutta, and was found dead in his bath-room—a very small aortic aneurysm having burst into the pericardium. An East Indian (?) medical subordinate died from an almost precisely similar cause. Prep. C 15 in the Grant Medical College collection is an 'aneurysm of the first portion of the arch of the aorta bursting into the pericardium.' Prep. 105, Series 5, in Sibthorpe's Catalogue shows an aneurysm of the ascending aorta, taken from a native who died from rupture of the sac into the pericardium. Prep. 65 is an aneurysm of the aorta, immediately above the valves and within the pericardium into which it burst. No. 59 is a small aneurysm of the aorta situated a little above one of the aortic sinuses. Death occurred by rupture of the sac into the pericardium by a very small opening.

I used to think that the progress of an aortic aneurysm through the chest was quite as capricious, as regards its direction, as that of a musket bullet. But the study, chiefly of cases observed in India, convinced me that, although the mode of expansion of a thoracic aneurysm is modified by various circumstances, its progress, which is generally to the surface, is ruled by definite laws. The following cases fairly display the principal modes in which aneurysms of the arch and descending aorta advance:

I collected a valuable series of plaster of Paris casts, taken by the late Mr. Bowser, from patients of mine, showing aneurysms of the ascending aorta, chiefly in natives, pointing forwards at and to the right of the sternum, causing absorption of bone, costal and clavicular cartilages, and, in two cases, opening by slough and allowing fatal hæmorrhage. A wealthy and rather noted Hindu gentleman, advanced in life, died with a large pulsating aneurysm about to point on the right of the sternum. I heard that, when his body burnt on the pyre, the sac burst throwing a column of blood into the air. It was manifest from the symptoms, although there was no autopsy, that the death of that truly eminent physician, William Twining, was caused by the opening of an aneurysm of the upper part of the ascending arch into the substance of the right lung. This occurred in a patient of my own.

A case which, as far as my reading and experience go, was unique,

occurred in the practice of my friend Dr. T. Watkins Wilson.¹ A man, *æt.* 28, born in India of English parents, a free liver, had suffered for 12 months from no other distress than a pain in the left deltoid muscle immediately under the acromion process, which prevented him from raising his arm. He then became subject to palpitation, difficulty in breathing, a feeling of constriction in the throat, and a pain to the left of the sternum. There was increased and extended dulness in the præcordial region. The first and second sounds were distinct, but feeble. Following the first, was a bruit most audible at the upper part of the sternum. The features were full, bloated and congested. Gradually respiration failed on the left side, and the whole of that side of the chest became dull. The thoracic suffering became very intense, and he died in a paroxysm of dyspnoea. It was found—

(1) That there was congenital transposition of the viscera, the liver lay on the left side, the spleen on the right.

(2) That an immense aneurysm, half filled with solid fibrine, occupied the usual site of the heart; the heart, with an adherent pericardium, was on the right side, displaced and thrust back behind the lung; the left lung was perfectly collapsed. Serum occupied the pleural cavity. The aneurysm appears to have arisen from the back part of the ascending aorta, and to have made way laterally to the left.

Aneurysms of the Cross of the Aorta.—A very delicate young officer, a younger son of one of the greatest literary men of this century, whose death was also from arterial disease, died from a large aneurysm of the upper part of the aortic arch. The sac would nearly have received two fists; I never saw such extensive arterial disease in a person under twenty-five years of age. The parts were so matted together that, as the specimen lay before me, it took me several minutes to understand it. He was in the Officers' Hospital, but was standing with his foot on a chair, talking and laughing, when he suddenly fell and expired.

In No. 2 of the 'I. A.,' I have detailed fully the very remarkable case of a ship's captain, aged 29, in whom I found, during life, evidence of the existence of an aneurysm of moderate size obstructing the three primitive branches of the aortic arch.

Under the heading of Diseases of the Organs of Respiration (p. 504), I shall have to report in detail the case of a Greek who had symptoms of acute laryngitis caused by a little aneurysm arising from the crown of the aortic arch attaching itself closely to the trachea in its upward course, and beginning to open into that air passage. I vainly attempted to save life by tracheotomy. I had to watch the sufferings, ending fatally in a few hours, of a poor European in whom an aneurysm was found to be making its way into the bronchus, nearly as happened in the case of Liston, the great surgeon; save that, in the latter, there was a pre-

¹ I. A., No. v. p. 362.

monitory gush, followed by a considerable interval of apparent health, in which I took a patient to him. He looked pallid ; but, as he held me by the button-hole, I thought that his was one of the noblest and most scholar-like faces I had ever looked upon. Mr. R. O'Shaughnessy published, in the 'Calcutta Medical and Physical Transactions,' a case in which aneurysm of the arch of the aorta was mistaken, during life, for aneurysm of the right carotid. I was asked to examine a young and well-built Queen's soldier, who had a pulsating swelling, as large as a hen's egg, at the root of the neck. It lay in the course of the left common carotid, and had much the appearance of an aneurysm of that vessel ; the application of a ligature distally was in contemplation. It, however, appeared to me that it was an aortic sac. My colleague, Dr. Chuckerbutty, had a patient, a youngish native man, in whom there was a huge pulsating sac arising from the aortic arch, and occupying the whole of the front of the neck. On placing my hand upon it, my first feeling was one of astonishment,—where was the larynx, of which there was no trace in front ? A projecting mass lay *just below the left angle of the lower jaw*. This was the larynx with its appendages, which had been pushed aside in the gradual upward progress of the aneurysm. Respiration was quite easy. I showed this to my class, saying that it appeared to give an additional physiological reason for the fact that our upper air passage is not an immoveable bony tube.

Aneurysms of the Descending Thoracic Aorta.—A much esteemed European officer, a well put together dark man, apparently under 35 years of age, was treated by the late Dr. Edward Goodeve, for profuse spitting of blood. There was extensive dullness under the left clavicle. At this spot, I think, a little moist crepitation was noticed. It was considered singular that there should be such extensive lung mischief, with excessive hæmorrhage, where all other signs of phthisis were absent. He went home, appeared to recover, and married. On his return to India, after several months' absence, he appeared well, and, I believe did not seek medical advice. I recollect to have seen him walking and leading his wife's horse—a considerable exertion in that climate. After sunset, on what was noted to have been a singularly hot day, he was about to walk out with his wife when he fell, bringing up blood, and expired. I carefully examined the diseased structure. An aneurysmal sac, of globular form, larger than a fist, proceeding from the front of the descending arch just as it turns, had burrowed into the substance of the upper lobe of the left lung, which it had not pushed away, but had almost completely destroyed by absorption. The amount of dullness and the profuse hæmorrhage were clearly evidence that the aneurysm had occupied that position and had bled largely before he went to England, and that Nature had made an attempt at cure, which was valid for many months.

Preparation 665 is from a European woman, a patient of mine. It displays an aneurysm of the extremity of the arch of the aorta *opening into the œsophagus*. Death did not result from hæmorrhage, although a little blood was spat up, but from pressure upon the trachea and consequent suffocation. The fatal attack of asphyxia was precipitated by a drinking bout. Many years ago, I saw, at the South London Medical Society, a fresh specimen in which the firm and even coagulum of an aortic aneurysm had completely removed an oval portion of the œsophagus, upwards of three inches in circumference. Death had not been caused by hæmorrhage, but the tumour encroached so much upon the passage as to cause dysphagia. It was clear that, if a probang had been used, as was then a frequent practice, the sponge would have entered the aneurysmal sac. I saw a very remarkable case of this kind in Calcutta. A European, rather past middle age, had difficulty in swallowing. There had not been hæmorrhage, but he was anæmic. Thus, his nose was quite pale, but there were large red vessels upon it, as if he had formerly been extremely florid. Some time after he came to me, the medical friend who had consulted me brought to my office an earthen vessel which appeared to contain more than two pints of blood which had come up at a gush. Not long before that time, I had been in the habit of making close examination of aneurysmal clots. With this blood, there was at least a handful of partially decolorised laminated clot which had unquestionably come from the sac of an aortic aneurysm, probably through a large opening in the œsophagus. The man was doubtless well cared for; there was no recurrence of hæmorrhage, and he went to an employment at Diamond Harbour, where he died about a year afterwards, it was said of some bowel complaint.

Prep. 109, Series 5, in the Madras collection, is an aneurysm of the descending aorta which burst into the œsophagus and into the anterior mediastinum. A large quantity of blood was brought up. The patient 'lived for two days after the sac gave way, the bleeding having stopped twice for a considerable interval either from failure of the circulation, or from a loose clot being forced into the opening by the rush of blood.' From a European soldier.

In Prep. 138, Appendix, p. 132, an aortic aneurysm, communicating with the œsophagus by the usual long oval opening, an inch long and half an inch wide, suddenly gave way, causing instant death.

I had long in my ward a middle-aged European who had been a soldier. His chief ailment was a harsh dry barking cough, which it was distressing to listen to. Examination *post-mortem* confirmed the diagnosis that this was due to the pressure of an aneurysmal sac on the left recurrent laryngeal nerve. He was so irritable and quarrelsome a person that I found it very difficult to keep him in the state of quietude which his condition demanded. After a time, the

cough abated, and he appeared to be mending, but he died from acute pneumonia, occasioned, I believe, by mechanical interference with the pneumogastric nerve.

An unhappy European sailor was for some time in my ward with an aneurysm of the descending thoracic aorta which had destroyed several ribs posteriorly on the left side, and formed a tumour so bulky that two hands could scarcely cover it. He went out, got drunk, fought, and was severely beaten, and returned to hospital for a time, still he did not die under our observation.

Preparation 678 represents a most interesting case which occurred in my ward. An elderly American master mariner became absolutely paraplegic only a few days before his death. The lower extremities had entirely lost sensibility and power of motion. He, however, declared that once, during the night, he found himself able to use his legs. I told my class that this state of things could only arise from one of three causes—1 and 2, strumous or malignant disease of the bodies of dorsal vertebræ. I considered that the man's occupation and grand physical development, together with the absence of any spinal projection, discountenanced struma; while his aspect and the fact that the cord had temporarily recovered its powers obliged us to put aside the second cause; whereas all indicia countenanced the opinion that—3—an aneurysm had destroyed the bodies of the vertebræ and was compressing the cord. It was found that an aneurysm of the thoracic aorta had largely destroyed the bodies of ten dorsal vertebræ. One of the lower bodies had become so completely absorbed as to expose the dura mater. Higher up, two bodies had been destroyed, also exposing the dura mater; and, still higher up, a portion of the body of another vertebra was disintegrated down to the same tunic. The huge sac was filled with a very dense coarsely-laminated coagulum. In one spot, the sac was about to open into the left lung. At first, it appeared probable that the idea of temporarily restored power of motion occurred in a dream; but he was positive that he had moved his legs. The truth of this appears quite possible, considering the manner in which the cord was compressed by the aneurysm, the volume of which was liable to be diminished from the quiet state of the circulation during sleep.

In 1845¹ I suggested that the chief general indications which it is necessary to fulfil, in conducting a rational plan of *Medical Treatment, in cases of aneurysm of the aorta*, are as follow: ²—

To reduce, as far as is practicable, the volume of the circulating fluid; but in such a manner as to lessen the proportion of its watery

¹ *London Medical Gazette* for August 29 of that year, p. 774.

² As revised in my treatise on the 'Treatment of Diseases of the Heart and of Aortic Aneurysm,' Calcutta, 1851.

constituents without diminishing the fibrine or producing debility in the system.

To render the circulation through the visceral arteries, general capillary system, principal venous trunks and lungs as free as possible.

To maintain the muscular power of the heart.

To encourage the deposition of *thin* layers of plastic coagulum within the aneurysmal sac.

These principles have always guided my practice. For many years, I used to tell my students that the greatest hope of prolonging his life that we can offer to the subject of aortic aneurysm is to afford him absolute REST, which he can best obtain in hospital, where I generally succeeded in keeping my patients with aneurysm until they died. Latterly, I employed the iodide of potassium treatment, but I was unable to attribute any effect to it. While it is, in these cases as in all others, the physician's obvious duty to prolong existence as much as possible, I have always admitted to myself that, in following this line of practice, we distinctly add to the sum of the patient's sufferings. For many years, my wards were very rarely without cases of aortic aneurysm. Only one of my patients died suddenly in hospital; but, as time advanced, the sacs increased in bulk and gradually made their way towards the surface and usually beyond it. Assuredly, if I were the subject of aortic aneurysm, I should accept, as a mercy, the visitation of sudden death vouchsafed as the alternative of months of suffering, while the mass, in growing, causes the destruction of every structure which stands in the way of its advance.

I saw only one case of *Aneurysm of the Common Carotid* in a native man. Death occurred suddenly by cerebral embolism.

Preparation 696 is 'an aneurysm of the right common carotid of a native, about an inch and a half above its origin from the innominate. Close to its entry into the cavity of the aneurysm, the lining membrane is slightly infiltrated with yellow atheromatous deposit. The tumour is globular in shape.'

Preparation 697 is from a Lascar, and displays aneurysm of the left common carotid at its bifurcation, extending along the external carotid. The common carotid was ligatured. The man lived a month. Death occurred from sloughing of the whole of the left side of the neck. The left cornea had sloughed.

Preparation 698 is one of great interest. It is from a man of 30, in whom, a year previously, the right carotid was tied by Professor Mott. The disease was aneurysm in the right orbit caused by an accident. He returned to England, and, the swelling not having disappeared, the left carotid was tied. He died anasarcaous, with albuminuria. The account of the condition of the vessels and of their irregular distribution is interesting.

Dr. John Shortt¹ tied the common carotid successfully in a native prisoner, *æt.* 34, for aneurysm of the external carotid.

Mr. Edward A. Birch ligatured the right common carotid artery with catgut in a Chumar, *æt.* 40, for aneurysm of the internal carotid. The operation was followed by headache, left hemiplegia, and mental confusion. The power of speech was lost and recovered. This was a typical case of those disorders of the cerebral circulation which I have shown, in the 36th volume of the 'London Medical Gazette' for 1845, to have resulted in many cases where the common carotid has been tied. He eventually made a good recovery.

Preparation C 1, in the Grant M. C. collection, represents another case of aneurysm of the left carotid artery. The disease seems to have commenced at the bifurcation. The seat of ligature is shown about an inch and a half from the origin of the vessel with a loose internal coagulum. The patient died a month after the operation. The larynx was ulcerated, and the cricoid cartilage necrosed. There were an abscess in the brain and a cavity in the upper part of the left lung.

Baboo Preonath Bose has reported² the case of a Hindoo hawker, *æt.* 32, suffering from a large aneurysm of the *right internal carotid*, who had the common carotid tied by Mr. S. Bowen Partridge. There were no unfavourable symptoms for seven days; then the right eye 'became suddenly and uniformly congested and, in the course of 48 hours, was entirely destroyed.' Hæmorrhage occurred on the separation of the ligature. On the thirteenth day, a ligature was placed around the innominate trunk.

The patient sank principally in consequence of pneumonia, a rather frequent result of operations for tying the arteries of the neck.

In the chapter on Diseases of the Nervous System, p. 405, I have spoken of the case of a Hindoo gentleman of high caste, in whom the presence of an aneurysm of a *cerebral artery, given off from the left internal carotid*, was evidenced by a remarkable systolic bruit.

Mr. A. H. Hughes successfully ligatured the *Subclavian artery* for axillary aneurysm in a native policeman, *æt.* 25.³

There is on record⁴ the remarkable case of an Indian major, *æt.* 33, who died suddenly from an aneurysm of the *Left Subclavian* opening into the left pleura.

A native woman died from *Aneurysm of the Abdominal Aorta* in my ward. She was extremely thin. There was considerable abdominal fulness; and a soft swelling, the strong pulsation of which was very apparent, showed a little below the left hypochondriac ridge. After death from rupture, the sac was found to be an immense one, having

¹ *I. A.*, No. xv. p. 269.

³ *I. M. G.*, vol. vii. p. 183.

² *I. A.*, No. xxvii. p. 332.

⁴ *I. A.*, No. iii. p. 461.

upon it several rounded diverticula, one of which had advanced to the abdominal wall, over the lesser curvature of the stomach, and had formed the pulsating tumour.

Preparations 679 to 683 are five typical cases of aneurysm of the abdominal aorta, but there is nothing to show whether the sufferers were Europeans or natives.

I do not think it needful to cite all of the many reported cases of aneurysm of the smaller arteries, in Europeans, which appear in our Indian literature, but I may allude to that of a young soldier in whom I saw my friend, Dr. Gordon Hardie, tie the *External Iliac* with perfect success, although there were marked evidences of a most extraordinary amount of disease in the poor fellow's arterial system generally; and to that of a well-conditioned looking European seafaring man who, while under treatment for other disease in my ward, noticed a swelling which I found to be *Popliteal Aneurysm*. Gangrene of the limb occurred, and he sank after amputation. I saw a nearly parallel case in Guy's Hospital.

In 1837¹ Mr. Richard O'Shaughnessy ligatured the *External Iliac* in a Calcutta Hindoo, *æt.* 40. There was a femoral aneurysm about the size of a large orange. The aneurysm appeared to be cured, although, when the case was reported, six weeks after the operation, the ligature had not come away.

There is a singularly interesting case² in which Dr. Parry successfully ligatured the *External Iliac Artery* in a European soldier in whom sloughing (of syphilitic origin) in the groin produced diffused aneurysm of the femoral.

Kenneth Mackinnon just mentions (p. 8) the case of a native in whom '*aneurysm of the External Iliac or one of its branches under-* a cure by sloughing.'

Sir Joseph Fayrer tied the *External Iliac* with success for femoral aneurysm in a West Indian negro, *æt.* 34, who had been several years in India.³

In a case where an aneurysm of the femoral artery ruptured at Poupart's ligament, the late Mr. Cutcliffe ligatured both the *External Iliac* and the *Femoral* arteries, using carbolised catgut.⁴

Many years ago, at Guy's Hospital, I watched the case of a man who, upon being admitted with an actively pulsating aneurysm of the femoral as large as an orange just below Poupart's ligament, was attacked with typhus. On his recovery from the fever in about six weeks, the aneurysm had undergone spontaneous cure.

I believe that the first surgeon who tied the femoral for *Popliteal*

¹ C. M. T., vol. viii. part 2, p. 223 of Appendix.

² Madras Quarterly Medical Journal.

³ I. A., No. xxvi. p. 146.

⁴ I. M. G., p. 29.

Aneurysm in India was Dr. Adam Macdougall, who operated, with complete success, in 1822, on a Hindoo ordnance driver.¹

Kenneth Mackinnon noticed that the same operation was performed successfully, at Madras, on a sepoy. This was probably the case reported by Mr. J. Lawrence.²

Sir Joseph Fayrer tied the femoral, at the superior third of the triangle, in a Burmese, aged 26, suffering from popliteal aneurysm. The whole of the sac sloughed away. Three days afterwards, profuse hæmorrhage occurred from the cavity which had contained the sac. The common femoral was then tied about an inch and a half higher up. Thenceforward, he made a good recovery.

Mr. F. F. Allen tried pressure and flexion in a native (Goorkah) corporal, aged 50, with popliteal aneurysm. He then tied the femoral in Scarpa's triangle with perfect success.³

Mr. C. P. Costello reported⁴ the case of a native soldier in whom he successfully treated an idiopathic popliteal aneurysm by combined pressure and flexion.

Surgeon Major P. Sinclair Laing cured popliteal aneurysm, in a European soldier of 30, who had served nearly seven years in India, by flexing the limb.⁵

Dr. John G. French⁶ gives the very interesting case of a Mussulman in whom he tied the artery, in Scarpa's space, for a false traumatic aneurysm of the femoral, wounded by duck-shot.

Dr. K. D. Ghose employed digital compression successfully for popliteal aneurysm in an up-country Hindoo aged 40.

The cases which I have quoted show clearly enough that aneurysm occurs in the natives of India nearly as it does in Europe; there, however, remains the not-easily solved question—Is arterial disease as frequent in India as it is in the United Kingdom? Kenneth Mackinnon, writing from *mofussil* (country) experience, stated it, as the result of his observation, that arterial disease is rare in India. Allan Webb, having read nearly all that had been published regarding disease in India, and writing with large experience of the Calcutta hospitals, held a contrary opinion. Writing earlier than these eminent authorities did,⁷ Richard O'Shaughnessy said that he was 'strongly persuaded that aneurysm is much more common in India, and that there are more lives lost by it among the natives, than is generally supposed; as,

¹ *C. M. T.*, vol. i. p. 199. Poor Macdougall's tomb is in our burial ground at Chittagong. It is said that, being on his way to that station, he died of cholera in his palanquin. As the bearers would not touch the body, they brought it, as it lay, to the station, where it was found that decomposition had so far advanced that a great pit was dug in which it was inhumed, undisturbed in the *palkee*.

² *C. M. T.*, vol. iv. p. 392.

³ *I. A.*, No. xix. p. 307.

⁴ *I. M. G.*, January 1, 1866.

⁵ *I. A.*, No. xviii. p. 261.

⁶ *I. M. G.* for November, 1866, p. 335.

⁷ In 1837, *C. M. T.*, vol. viii. part 2, Appendix, p. 223.

from their total ignorance of the formidable nature of the disease, they apply to the native quacks in the bazaars, who, taking the swellings for simple abscesses, puncture them.' I find that, in 1881, there were only 6 admissions for aneurysm and 3 deaths out of an average strength of 114,612 sepoys; while, during the same period, the European army, with a strength averaging 58,728, had 9 deaths from aortic aneurysm, and sent away 9 cases of aneurysm invalided or for change. The result of my own experience is that aneurysm is a rare disease in the natives of India, although I saw many cases in Calcutta, the largest city.

Cases of *Arterial Plugging* are noticed in the chapter on Malarial Cachexia, p. 255. I never saw or heard of a case of *Senile Gangrene* in a native. There are not many very aged Europeans in India. I saw only one case, noticed at p. 464, of this disease in an Englishman.

DISEASES OF THE VENOUS SYSTEM.

Mr. Charles N. Macnamara has alluded to the case of a European soldier, the subject of general fatty disease, in whom '*the Superior Vena Cava was ruptured* just at its junction with the auricle.'¹ A soft yellowish jagged spot, about the size of an eight anna piece (shilling), appeared in the descending cava just at the point where that vessel enters the heart. There was, at the centre of this spot, a small opening through which fatal hæmorrhage had occurred. The walls of the vein, in this locality, had entirely lost their normal structure, being replaced by fat. The remainder of the vessel, as also the coats of the aorta, the heart, liver, and kidneys, were all in a state of fatty degeneration.

Dr. John Shortt gives² the case of a native Christian butler, aged 50, a fat man, said to be intemperate, who fell suddenly, and was fifteen minutes dying. 'A circular rent, about the size of a shilling, was found in the superior cava within the pericardial sac, and near its termination in the upper part of the right auricle. Half an inch of the cava round the rent had its serous coat destroyed, and the surrounding tissues were infiltrated with blood.'

Mr. George Harper has given³ the case of a European riding-master, aged 45, an unhealthy man of temperate habits, in India for 25 consecutive years. His horse becoming restive and making a sudden twist round, he was obliged to use great muscular exertion of the lower extremities to enable him to keep his seat. Immediately after this, he felt the right leg stiff and uneasy, and the whole of that extremity became highly ecchymosed from the ilium to the ankle. He

¹ *I. A.*, No. v. p. 182.

² *Ibid.* No. xv. p. 186.

³ *Ibid.* No. xv. p. 256.

survived the accident 41 days, during which his health steadily declined. A circumscribed strongly pulsating tumour, nearly the size of a goose's egg, was felt immediately above and opposite the umbilicus, over which there existed a distinct bruit. It was suspected that aneurysm of the abdominal aorta or common iliac existed. He suffered greatly from constant lumbar and abdominal pain, and from attacks of dyspnœa; and, latterly, there was colliquative diarrhœa. No arterial lesion was discovered *post mortem*, but the *Vena Cava* was *enormously distended* for about two inches before passing through the hepatic fissure, and was filled with a large clot of blood. The rectus femoris was ruptured, and it and nearly all the muscles of the thigh and leg had large clots in and about them.

It is not stated whether Prep. 702, in the C. M. C. M., is from a native or a European. There is '*Obliteration (embolism) of the Inferior Vena Cava* opposite the termination of the renal veins.' At this point, the vein is three times its natural thickness. The tumour is about an inch and a quarter across and two inches in length. 'Above this, the vessel is thin, atrophied and cord-like.' Nothing is said regarding the manner in which the collateral circulation was carried on.

Two cases of obstruction to the inferior cava have occurred within my personal knowledge. In one of these, I dissected the tissues of the enormously enlarged external epigastric veins, and found that, unlike any other integumentary veins, these generally small and unimportant vessels are constructed precisely as the inferior cava is, evidently with a view to enable them to act vicariously for that trunk when it is unable to do its own work.'¹ For the other case, see Phlegmasia Dolens in Diseases of Pregnancy.

For observations on *Venous Plugging* in Malarial Cachexia, see p. 254.

Varix is not common among the civil population of India. Throughout my India career, I generally looked the natives well over wherever I went, and thus often noticed remarkable evidences of disease. Among the hundreds of thousands of bare legs which I must thus have glanced at, I can only recall to mind one very striking example of varicose veins, in the leg of an Ooriah *palkee*-bearer.

The Returns for 1883 give the following admissions for Varicose Veins: European soldiers, 71; Native troops, 41; Prisoners, 3.

DISEASES OF THE RESPIRATORY SYSTEM.

For *Influenza* in India, see p. 111. For *Croup* and *Diphtheria*, p. 114. For *Pertussis*, p. 109.

Hay Asthma, or rather *Hay Fever* (*vide* p. 242). In the first

¹ *London Medical Gazette*, August 8, 1845.

published draft of this chapter, I said that this affection does not appear to have been observed in India.¹ Upon this, my friend Dr. David Boyes-Smith, of Netley, wrote: 'I knew a surgeon at Meerut, many years ago, who suffered from hay fever very severely at that station.' I have alluded to the case of an aged civilian, long retired, who told me that he was subject to hay fever before he went to India, and lost it there. In one of his valuable notes on Indian disease, the late Dr. Hogg wrote: 'At Umballa, quite recently, a young officer, a martyr to hay fever at home, wondered why he enjoyed such entire immunity in India.' A lady, a patient of my own, first went to India in middle age; she never had hay fever until she returned to England. On going back to India, the affection left her. Four other ladies of my acquaintance, three of whom were born in India, have become painfully subject to the malady in England. All suffer from coryza, one of them is asthmatic.

Asphyxia.—At pp. 645-652 of my 'Medical Jurisprudence for India' I combated the then generally accepted opinion that the essential and invariable characteristic evidence of death by asphyxia is the accumulation of blood in the right heart and in the lungs. I adduced evidence in proof of the fact, which I felt confident would thenceforward be generally accepted, that, although in many cases of asphyxia the muscular power of the right ventricle is overcome, and its cavity is found gorged after death, there is a set of instances in which, the action of the heart continuing after the cessation of respiration, the right ventricle is found well contracted and nearly empty, the lungs being congested in an extreme degree; and I urged that, unless this fact be admitted, it may, in certain cases, be found difficult to substantiate our opinion that death has resulted from asphyxia.

After my work appeared, Dr. Alfred Taylor added his own authority to the principles which I then advanced.

I subsequently noted the following characteristic observation, by Dr. Packard, of Philadelphia.² It is given as 'an instance of pure suffocation by hanging.' A very strongly-built and muscular man, *æt.* 37, hanged himself to a banister with a piece of picture-cord wrapped with soft cotton to give it bulk; it was so short that he could scarcely have had more than six inches fall; he could not have been hanging more than a very few minutes when he was discovered, but death had already occurred. In the autopsy, 48 hours after death, the body having been in ice, it was found that there was strong *rigor mortis*; the upper surface was very pale, but hypostatic congestion of florid hue was present except where the body had pressed on the bottom of the box. There was considerable cerebral congestion, with some ecchymosis and slight extravasation of blood. 'Lungs, healthy and

¹ Vol. i. for 1884, p. 78.

² *American Journal of Medical Science* for July 1870.

deeply congested, especially at their posterior parts. Heart, well-developed, *strongly contracted and empty.*' [The italics are mine.—N. C.] 'On its surface, a somewhat unusual amount of fat; valves all healthy. Blood dark-coloured and perfectly fluid. Abdominal viscera healthy. Liver full of blood, although its surface was rather paler than normal.'

Edema Glottidis, in syphilitic disease. In a strong European, suffocating apparently from this cause, I waited as long as possible by the bedside, it being the rule of our hospital that surgical cases, occurring in the physicians' wards, should be made over to the surgeon of the day. At last, the patient fell over, and I opened the trachea. I could scarcely adjust the canula when, as frequently happens, the patient, tired out by long-continued dyspnoea and unrest, fell asleep. He recovered without a bad symptom.

Polypus.—During convalescence in the above case, the canula, having been long kept in, brought away a growth of mucous membrane about as large as a pea, which had not produced any evident obstruction.

Laryngitis.—Sir Joseph Fayrer has placed on record¹ the case of a delicate-looking native man, *æt.* about 28, in whom he performed tracheotomy at the moment when the patient was on the point of death from acute laryngitis. Writers on tracheotomy will find that the details of this case are replete with valuable suggestions.

I had an undoubted case of *Acute Idiopathic Laryngitis* in a youngish native woman under sentence of death in the jail at Howrah. It yielded to treatment without much difficulty. I had a case of laryngitis, and more, in the person of a very powerful Greek, whose strength had qualified him to keep a drinking shop in Lall Bazar, the chief resort of sailors in Calcutta. Overlooking the fact that he had become old and fat, this man had a fight with a sailor stronger than himself, was very severely beaten, and fell into ill-health. He was admitted to my ward with very characteristic symptoms of acute laryngitis. Respiration was markedly more interfered with than it usually is in simple laryngitis; and he, with great difficulty, got rid of large quantities of thin mucous froth. I was with him when he suffocated, and did not lose a moment in opening the air passage, but without effect. We found that the laryngitis was caused by an aneurysm, of about the size and flattened rounded shape of a child's toy watch, which, arising from the cross of the aorta, grew upwards, and was closely adherent to the bifurcation of the trachea, where it was about to open, having raised a swelling of the mucous membrane very like a small red currant. A case, occurring in a native, very closely similar to this, occurred three years ago in the Madras General Hospital.² He had suffered for about twenty days from cough with

¹ *I. A.*, No. 3, p. 427.

² *Annual Report of Civil Hospitals, &c.* for 1883.

pain in the chest, but the difficulty in breathing became urgent—expiration being chiefly affected—only on the day previous to his admission, since when no food was taken. Nothing could be made out on auscultation. The pulse was thready and rapid, skin cold and clammy, and the expression anxious. Tracheotomy was at once performed with much benefit, but he died rather suddenly twelve days after, in the act of coughing up blood. An aneurysm of the arch of the aorta had burst, by a minute aperture, into the trachea. Under the head of *Parotitis* ¹ I have described the cases of two little European brothers who were attacked, one fatally, with acute laryngitis [diphtheria?] in the course of that disease.

While in India, I regarded uncomplicated idiopathic laryngitis as one of the rarest diseases of that country. Except in one very questionable specimen, Prep. 719, the condition is not represented in Ewart's Catalogue of our Medical College Museum. The returns of the Sanitary Commissioner, for the four years ending 1883, however, display what must be regarded as a very extraordinary prevalence of this disease. Thus—

LARYNGITIS.

1880.

European Soldiers	4 deaths.
„ Women	3 admissions.
Native Troops	317 admissions, 56 deaths.
Prisoners	46 admissions, 16 deaths.

1881.

European Soldiers	3 deaths, 4 invalided.
„ Women	1 admission.
Native Troops	105 admissions, 8 deaths.
Prisoners	38 admissions, 8 deaths.

1882.

No deaths from laryngitis among the European soldiers, of whom 2 were invalided for this disease.

European Women	1 admission, 1 death.
Native Troops	90 admissions, 5 deaths.
Prisoners	22 admissions, 2 deaths.

1883.

No death from laryngitis among the European soldiers; one man was invalided.

No case occurred among the European women.

Native Troops	91 admissions, 2 deaths.
Prisoners	38 admissions, 4 deaths.

As croup and laryngitis are taken together in the four years' returns of European children, I do not cite the figures.

¹ Page 110.

At first sight, it might appear that this disease was laryngeal diphtheria, prevailing in Upper India not only among the sepoys on field-service, but also among the inmates of the jails; but this supposition is disallowed by the fact that the returns of the four years give only four admissions for diphtheria among the native troops and prisoners in the three Presidencies, and these only in the years 1880 and 1883.

In 1883, the designation of the disease, as it occurred among the native soldiers and prisoners, is altered from 'Laryngitis' to 'Laryngitis and Laryngeal Catarrh.' This appears to be indicative of a change of type in the malady.

As I think that these statistics can only be satisfactorily explained by medical officers in whose hospitals this disease has been prevalent, I shall refrain from merely speculative comment upon them.

I saw a case of Sir Joseph Fayrer's in which [syphilitic?] *Necrosis of the Thyroid Cartilage* proved fatal by pyæmic deposits in the lungs, &c. In July 1881, Dr. McLeod noticed, at the Calcutta Medical Society, a case of necrosis of the left ala of the thyroid cartilage in a case of chronic laryngitis, in which laryngo-tracheotomy was performed. The necrosed fragment occupied a gangrenous abscess cavity, which communicated with the cavity of the larynx. The patient died of lung disease after a lingering illness.

At the same time, Baboo O. L. Mookerjee exhibited a preparation of *Necrosis of the Cricoid Cartilage* from a Hindoo who was admitted moribund, suffering from very urgent dyspnœa. He was unable to speak, but pointed to his throat as the seat of his malady. The cricoid was in a state of necrosis and was surrounded by an abscess. The lungs were hepatised. Note that all of these patients died at the lungs.

In November, the same gentleman reported another very similar case. A Hindoo was admitted with double pneumonia, from which he recovered. Sponginess of the gums was subsequently noticed. In about three weeks, pain in the throat was complained of, the temperature rose, the voice became laryngeal, and dyspnœa set in. He died a week later. The cricoid cartilage was bare, and lay in a sloughy abscess.

I sent Preparation 727 to the Medical College Museum—'*Abscess between the Thyroid and Cricoid Cartilages*, and œdema of the laryngeal mucous membrane of the right side, also implicating the glottis.'

Fracture of the Larynx.—In my 'Medical Jurisprudence' (page 584) I have noticed a case, mentioned to me by the late Dr. Duncan Stewart, in which an aged woman, who was found dead in her bed, had one of the wings of an ossified thyroid cartilage broken and forced inwards, causing suffocation.

Preparation 713 in the Calcutta Medical College Museum represents 'Ossification of the epiglottis and all the cartilages of the larynx, and fracture of the anterior portion of the ossified cricoid cartilage.'

The rings of the trachea are also ossified, and there is calcareous deposition in the elastic ligamentous material connecting the rings to each other; from an aged Armenian woman.'

A very interesting case of *Mechanical Injury to the Upper Air-passages* occurred in the practice of Mr. Maitland at the Madras General Hospital.¹ A native, having been throttled by another man in a quarrel, was brought in suffering from very difficult respiration, the inspiratory act being accompanied by collapse of the intercostal spaces and abdomen. Râles were heard all over both lungs, and there was bleeding at the back of the throat. It was impossible to ascertain what injury had been caused. Tracheotomy was performed immediately, and he made a good recovery.

Foreign Bodies in the Air Passages.

M. Nepveu has reported² the case, forwarded by a military surgeon from Algeria, of a robust Arab, who stated that, for a fortnight, he had had a leech in his throat, which caused him to spit blood every ten minutes, and induced dysphagia with violent cough, without much dyspnœa. The laryngoscope displayed the leech stretching from the summit of an arytenoid cartilage to the base of the epiglottis, leaving however, the glottis pretty free. It was extracted living, by means of the Cusco forceps, and all the symptoms soon disappeared. M. Nepveu refers to six other similar cases which are on record.

Dr. Webb relates the case of a European girl, aged 12, who suffered in an extreme degree from splenic cachexia. She died suddenly, in a fit of coughing. A large worm was found in the pharynx, pressing down the epiglottis, which was very red from irritation. (Page 153, 'Pathologia Indica.')

A similar accident occurred in Madras in 1872.³ A horse-keeper, in hospital for tetanus caused by the bite of a horse, was talking in monosyllables to the nurse, when there was a violent spasm accompanied by extreme contraction of the muscles of the back, lividity of the face, fixed and protruding eye-balls, firm contraction of the flexors of the hands, and cessation of respiration. The spasm terminated as suddenly as it had commenced, and with it the life of the patient. A round worm was removed from the mouth about a quarter of an hour before death. It occurred to Mr. Brockman that probably a worm had entered the wind-pipe. There was found, lodged there, a round worm about seven inches long. Its head extended far into the left bronchus. Its tail lay just below the true vocal cords.

Preparation 737 in our Calcutta Museum shows three round worms

¹ *Report of Civil Hospitals and Dispensaries*, p. 72.

² To the Paris Société de Chirurgie, cited in the *M. T.*, vol. i. for 1884, p. 46³.

³ *Madras Monthly Journal of Medical Science* for March of that year, p. 190.

in the bronchial tubes. 'There is an ulcerated communication between the œsophagus and the trachea, near its bifurcation, through which the entozoa have gained access to the air passages.'

At page 619 of my 'Medical Jurisprudence for India' will be found a case which I saw admitted in 1865 to the surgical wards at the Calcutta Medical College Hospital. A native boy of 4 appears to have placed a Coie fish, which is very tenacious of life out of water, in his mouth. In its struggles, its head passed the glottis and caused suffocation, as all attempts to draw it out were prevented by the catching of its gill-plates, anchor-wise, below the vocal cords. Other similar cases of laryngeal choking by fish will be found in the above-named work.

The following appears in the 'Lady's Magazine' for September, 1771, p. 143: 'On the 5th of this month, as a fisherman of Whitstable was pulling a small sole out of the mesh of the net with his teeth, it slipped into his throat. His companions saw the tail, but could not get hold of it, and, the fish insinuating itself into his windpipe, he was suffocated in a few minutes.'

The following case gains importance from the fact that the condition of parts was examined after death. The body of a Mahommedan, *æt. cir.* 22 years, was brought to an outpost of Chittagong. He was supposed to have died from a *Koi* fish having stuck in his throat. Being out fishing, he caught hold of the fish, and, on finding a second, tried to secure the first with his teeth and it slipped into his throat. He and his companions tried to get it out, but it worked its way further in, and he died in an hour from suffocation. The fish (*kholso*) was found plugging the larynx and trachea, the tail being within the vocal cords. It was about three inches long and one and a half inch broad. The mucous membrane of the larynx and trachea were deeply congested. Froth and blood filled the bifurcation of the trachea.¹

A case has lately been reported² in which the death of a boy, *æt.* 15 months, was caused by the presence of a *shrimp's* head at the bifurcation of the trachea. Mr. Macnamara³ was successful in removing a *cowrie shell* by tracheotomy in a native child *æt.* 4½ years.

For cases of impaction of living fish in the gullet, see *Foreign Bodies in the Pharynx*.

Prep. 32, Series 6, in the museum at Madras, is Larynx of a child with a tamarind seed impacted immediately below the rima glottidis. Death took place in about 25 minutes.

Dr. J. Clarke details⁴ the case of a girl 12 months old, suddenly attacked with difficulty in breathing. She was apparently dying that night, when tracheotomy was performed. Free respiration followed the

¹ Assistant-Surgeon Bholanath Pal, of Cox's Bazar, *I. M. G.* for 1885.

² *M. T.* for September 5, 1885.

³ *I. M. G.*, vol. viii. p. 295.

⁴ *I. M. G.* for March 1885.

introduction of a vulcanite tube ; but, when milk was given, although it was swallowed eagerly, it came out beside the trachea-tube and caused a good deal of spasm and cough. Death, from exhaustion, on the third day. At the junction of pharynx and œsophagus, was a swelling of about the size of a small egg. This burst in removal, discharging a quantity of pus. It was formed of the walls of the pharynx and œsophagus much thickened and infiltrated. In this, was a piece of tin about an inch square, with a string attached. It was bent on itself at the centre and was firmly attached to the anterior wall of the pharynx and back of the larynx. The back of the bend was directed backwards, towards the posterior wall of the pharynx ; and the two sides, with four sharp corners, lay forwards towards the larynx. The right side of the tin had ulcerated through into the larynx, and projected a little to the right of the middle line, explaining the flow of milk from the trachea. The left side had made a hole in the wall of the pharynx, but seemed to fill it up, preventing infiltration of the deep tissues of the neck.

Bronchial Glands.—Preparation 743. ‘Tuberculosis of the bronchial and cervical glands, and obliteration of the left pulmonary artery from pressure caused by a bronchial gland greatly enlarged by strumous infiltration. The yellow tubercular matter is also seen in the small portion of lung preserved.

Preparation 744. Part of the trachea, bronchial tubes, and lungs. There is a ragged orifice, as large as a sixpence, at the bifurcation, and a smaller one in the right bronchus, caused by the breaking down of bronchial glands infiltrated with tubercular material.

I met with a case in which a calcareous mass, which had formed in a tubercular bronchial gland, was making its way through an ulcerated opening, large enough to admit the point of the little finger, in the trachea, just at its bifurcation.

Foreign Body in the Left Bronchus.—Two young medical men, whom I shall designate as T. and S., were passing some stone-masons at work in Moorgate Street. T., while laughing heartily, felt something strike his soft palate sharply. The pain was so great as to confuse him, but he immediately spat some blood and felt certain that a fragment of stone had passed into his left bronchus. T. and S. entered the Indian Service ; S. died early of phthisis. T. was, at about the same time, suffering from most distressing symptoms which much resembled those of phthisis. He was much emaciated, was very weak, and had cough with expectoration. I attended him for some time. There was considerable subclavicular dulness on the left side, with small moist sounds. He was positive that the stone was still lodged in the left bronchus. There was no sound to prove this ; and, if a stone was really there, the case was exceptional, as foreign bodies usually lodge in the right bronchus. I counter-irritated very strongly. His health

improved, and he accepted an appointment up country. He did not live many months. I was unable to obtain particulars of his last illness. The body was not examined.

I was at Guy's Hospital when Mr. Key was engaged upon the most interesting case of Mr. Brunel, who had a half-sovereign lodged in his *right bronchus*; and I was present at some of his trials, upon the dead body, to reach a similar coin with curved forceps through an opening in the trachea, &c. There was, nearly at the same time, a poor fellow in the wards, in whom there was clear evidence of the presence of a sixpence in the right bronchus. He was a turnpike-keeper in the west of England, and met with the accident in shouting to a driver while the coin was in his mouth. Dr. T. A. Wise records¹ a remarkable, but not perfectly demonstrative, case of an English gentleman serving in India, in which severe pulmonary symptoms appear to have been excited by the presence of a fish-bone in some part of the air-passages—apparently the right bronchus.

At pages 617–18 of my 'Medical Jurisprudence for India' will be found various Indian cases of *suffocation from the presence of foreign bodies in the air-passages*. In addition to these, Surgeon-Major William Curran has alluded, in his interesting paper on 'Sudden Death,'² to three cases of soldiers in whom vomiting, while they were lying drunk, caused suffocation.

In October 1881, it was reported in the London papers that a perfumer, while sitting with some friends, suddenly quitted them. He was found insensible in the garden. He was taken to the Middlesex Hospital, but was found to be dead. There were evidences of suffocation in the trachea and lungs. It was believed that he had retched, had taken a sharp inspiration, and had drawn the vomited matter into the trachea.

Water and foreign bodies may enter the air-passages in submersion and drowning—vide my 'Medical Jurisprudence,' pp. 643–44, especially an important case of a young boy found drowned in a tank. I noticed small portions of green vegetable matter in the fauces, larynx, and trachea, and the right bronchus was almost completely filled with a portion of an aquatic weed so large that it was astonishing how such a body could pass the rima. No weed of the kind grew in the tank where the body was found; but it was proved that the boy's body had been discovered by a woman in a tank near his home, in which the weed grew abundantly. This female had conveyed the corpse to the more distant tank, which belonged to a person against whom she bore a grudge.

At pages 223–24 of the same work, I have cited cases, by Dr. James Wise and others, of *Aphonia*, resulting from Indian hemp intoxication in confirmed *gunjah-smokers*.

¹ C. M. T., vol. ix. p. 162.

² I. A., No. xxxii. p. 361.

Glottidis Spasmus.—See Tracheotomy in 'Hydrophobia,' p. 429.

Death from Spasm of the Glottis in the First Paroxysm of Tetanus.—Dr. A. Webb told me that a little boy, at La Martinière School, was having an ulcer on his leg dressed when he fell back in opisthotonos, and immediately died. At the time of the trial of Palmer, it was rumoured that a negro servant, in Edinburgh, cut his hand with a broken plate while waiting at table, fell, and died, in a tetanic spasm, in a quarter of an hour; but a record of the case could not be discovered. I have lately found this case, cited by Ballingall, 'Military Surgery,' p. 255.

Tracheotomy (*vide* p. 120).—I opened the air passage six times in India: in three Europeans, in two natives, and in an East Indian infant; in two cases of diphtheria, and in one each of syphilitic laryngeal disease, whooping-cough, aortic aneurysm, and hydrophobia. Two of these cases did well, as far as the operation was concerned. In five, the opening was made at the instant of apparent death; in one of these the patient recovered. Here I must repeat a surgical platitude, so important have I found it in practice: *the canula can hardly be too large*. I saw a case many years ago in London, in which it was clear that suffocation had been promoted by the use of a small canula. The fatal error in more than one ingenious instrument, strongly recommended by its inventor, has been that it did not leave an opening equal in size to the rima glottidis. The external incision should be free. It is to be borne in mind that the veins of the neck, from which troublesome hæmorrhage is to be apprehended, are most engorged when we operate late. Ample deliberation, in making the incisions, is, however, always allowable. If the patient be dead, a few seconds can make no difference; if not, he will either recover immediately or may be restored by Sylvesterism. In one of my cases, where the neck was enormously fat, I hurried, and there was great venous hæmorrhage. Mr. R. W. Parker's advice to remove the exudation in tracheotomy for diphtheria should never be lost sight of.

Bronchial Catarrh and Bronchitis (*vide* 'Influenza,' page 111) are common throughout India whenever the damp air becomes chilly, especially at the commencement of the rains, and still more so at the setting-in of the cold season. Then asthmatics, the phthisical, and the aged Europeans and natives, get severe chronic bronchitis.

Under the head of Influenza, I have given some account of the *Capillary Bronchitis*, which was extremely and very destructively prevalent among children in Bengal in 1828.¹

Dr. Macpherson, of Bauliah, remarked, with regard to the climatic condition at that time, that the wind, which during the rainy months in Bengal blows almost invariably from the east or south-east, was then (August and September) generally southerly, '*with hot sultry days and strong gales at night*.' The sentence which I have here

¹ See also p. 236.

italicised tells us as much, in explanation of the causation of nine-tenths of the acute disease which occurs in India, as many elaborate treatises do.

One morning, when I was unable to visit my wards, a European was brought in from a burning house. He was suffocating from a draught of hot air or gas, against which his epiglottis did not close, drawn into his lungs, probably when shouting. My colleague bled him freely to relieve the terrible asphyxiating congestion, but he rapidly sank.

I know of only two cases of *Bronchitis with tube casts in India*, both in medical officers, who upon retirement have enjoyed active health and are now aged men. I think that, in these cases, there was also malarial asthma.

For *Malarial Spasmodic Asthma*, see remarks in the section on 'Malarial Cachexia,' page 236.

Dr. Hogg¹ found that, in the jails of Lower Bengal, 129 died out of 3,415 treated for asthma. But what diseases were here comprehended under the designation 'Asthma'?

Pneumonia.

In speaking of *Idiopathic Pneumonia*, in the section on Malarial Cachexia,² I have mentioned that I saw very few cases during my long experience in civil practice in Lower Bengal. A very observant man, Mr. J. Murray, writing in the Bombay Presidency,³ observed that, during eight years, he had not met with a single case of pneumonia in a European, or with an idiopathic case of the disease among natives.

Morehead shows that 'Pneumonia is a rare disease in Europeans in Bombay'; but that, 'in natives, pneumonia of asthenic type is common.'

When pneumonia does occur in Bengal Proper, it is very apt to attack the *upper* lobes of the lungs of ill-fed cachectic subjects.

Hugh Macpherson found that, in the eight years 1853-54, inflammation of the lungs gave half per cent. of admissions in the European army serving in Bengal, and fifteen and a half per cent. of deaths to treated; which, compared with Col. Tulloch's columns, shows this disease to have been only half as common, but twice as fatal to those attacked, in India as in England.

The Sanitary Commissioner's Returns give the following statistics of Pneumonia throughout the three Presidencies:—

1880.				
European Soldiers				143 died, 3 invalided.
" Women				12 admitted, 4 died.
" Children				12 admitted, 7 died.
Native Troops				3,370 admitted, 1,222 died.
" Prisoners				1,669 admitted, 1,222 died.

¹ *I. M. G.* for 1875, p. 314.

² Page 234.

³ *I. M. J.*, vol. ix., O. S., p. 253.

1881.

European Soldiers	86 died, 10 invalided.
Women	6 admitted, 1 died.
,, Children	16 admitted, 9 died.
Native Troops	2,007 admitted, 593 died.
,, Prisoners	1,082 admitted, 398 died.

1882.

European Soldiers	1 died, 9 invalided.
,, Women	4 admitted, 1 died.
,, Children	14 admitted.
Native Troops	1,560 admitted, 384 died.
,, Prisoners	1,018 admitted, 349 died.

In the above years, the average strength was : European Soldiers, 58,571 ; European Women, 3,741 ; European Children, 6,784 ; Native Troops, 118,630 ; Native Prisoners, 100,556.

Just before going to press, I have obtained the following statistics for the year 1883 :—

European Soldiers	32 died, 9 invalided.
,, Women	9 admitted, 1 died.
,, Children	9 admitted, 3 died.
Native Troops	1,541 admitted, 380 died.
,, Prisoners	1,084 admitted, 351 died.

Epidemic Pneumonia in India.

At page 106* of Webb's 'Pathologia Indica' is a report by Dr. Green, which shows that, between February 1844, and May of the following year, an epidemic or endemic of pneumonia, frequently complicated with pleurisy, attacked the prisoners at Midnapore, in Bengal Proper, which stands, on a laterite soil, outside the Gangetic delta. In the sixteen months under report, there occurred a total of 175 cases of pneumonia, 14 of phthisis, and 10 of bronchitis. In the several months, the cases of pneumonia occurred in the following numbers: 0, 2, 7, 2, 7, 1, 3, 15 (September, a very hot month), 22, 13, 37 (December, the coldest month), 24, 19, 21 (March, hot weather), 8, 4. Dr. Green's paper is well deserving of study, especially in connection with Mackinnon's remark upon epidemic pleurisy. I do not think that any such outbreak has occurred in a jail of *Lower Bengal*, within my experience, since 1848. Webb cites (p. 100a) a report by Dr. Edward Goodeve upon the prevalence, at Cawnpore, of pneumonia and of other pulmonary disease simultaneously with the Midnapore outbreak in 1844-45. Writing in 1848 of a low type of pneumonia, Webb says, 'About two years ago this form of pneumonia, terminating in ["pulmonary"] œdema, was epidemic at Delhi, and very fatal.' Mackinnon says, writing at Cawnpore in 1848, 'Pneumonia is common in this

part of India, at least as a complication in fever, and as an idiopathic affection.' My own sensations, while in the keen northerly winter air on two nights at the Patna and Allahabad railway stations, and again, on arriving before dawn, also in a cold season, at Cawnpore, fully enable me to understand that the cold-weather climate of the North-Western Provinces, following abruptly a season of great heat, tends to excite acute pulmonary disease.

With reference to Mackinnon's remark upon the occurrence, in the North-Western Provinces, of pneumonia as a complication of fever, I have already cited, in the section on Malarial Cachexia (p. 235), a striking observation on this subject by Mr. Oldham. Of 103 cases of pneumonia in natives, treated by Morehead, 76 were of primary pneumonia and 27 of pneumonia complicating intermittent (4) or remittent (23) fever.

Pneumonia has always been recognised, in its epidemic form, as influenza; and we have several records of the endemic and epidemic occurrence of this disease in old times. Of late, its tendency to assume the epidemic character has been rather closely investigated. Dr. G. B. Longstaff has published¹ some important suggestions in reply to the question 'Phthisis, Bronchitis, and Pneumonia: are they Epidemic Diseases?' Mr. Henry E. Armstrong, Medical Officer of Health, has observed² that the increased prevalence of pneumonia in Newcastle, during the third quarter of 1883, was much greater in certain districts of that city than in others; thus, in Westgate, the deaths were equal to an increase of 85 per cent.; whereas, in St. Andrew's and St. Nicholas's, there was a decrease from this cause. This circumstance, he held, cannot be explained on the ordinary grounds of coldness of season, &c., but may be due to local circumstances, possibly in the form of sanitary defects. He adds that it is considered by some that 'certain kinds of pneumonia are the direct expression of the effects of zymotic poison rather than a condition of simple inflammation of the lungs; and that the relatively increased prevalence of the disease in certain parts of Newcastle would appear to support this view.'

I do not know how it may have been of late years in London, but, fifty years ago, *herpes labialis* was a very frequent attendant of pneumonia; and, quite within my recollection, pneumonia attended with 'typhous deposit,' occasioning extensive lung consolidation, was so marked a feature of the great London outbreak of maculated typhus of 1838 that the pulmonic complication was, to say the least, the most prominent feature in the disease.

A most useful work will be accomplished by those who may succeed in tracing the geographical spread of the very marked epidemic of pneumonia which prevailed in New York and, at least, in England,

¹ *Transactions of the Epidemiological Society of London*, N. S., vol. ii. p. 119.

² *Report on the Recently Increased Death-rate of Newcastle-upon-Tyne, 1883.*

Scotland, and Ireland, late in the spring of 1885. I noted the following, in a South African report for August 1885,—‘Inflammation of the lungs has been very bad at Kimberley—quite an epidemic, in fact; and it is said to have proved more fatal to the white population than the small-pox last year.’ There was a marked decrease when the report was made.

Dr. J. W. Moore reports¹ that, ‘in 1883–84, of 74 cases of croupous pneumonia in Cork Street Hospital, 9 proved fatal; and, in the epidemic year, 1884–85, of 101 cases admitted, there was a mortality of 25 per cent.

The following scattered notes suggest the advisability of compiling from the records a full history of the *Epidemics of Pneumonia* which have occurred in Northern India within the past fifty years.

Mr. R. H. A. Hunter, of H.M. 57th Regiment, gave² the following account of an outbreak which occurred in the Afghan war in 1839. It appears that very little pulmonary disease occurred until the army, having crossed the Indus, penetrated the high mountain districts of Afghanistan. At the termination of the first campaign, on the descent of the troops through the different passes, what appears to have been regarded as fever with pulmonary complication for a short time assumed a most malignant character, and, in a milder form, followed the several corps for months afterwards. Not only did the 2nd and 17th Regiments suffer much from the time of their emergence from the Gundana Pass in the beginning of December 1839 (see ‘Transactions of the Bombay Medical and Physical Society,’ vol. iii.), but so also did the Bengal column on their descent into Tezeen. The most destructive epidemic of this kind which came to the reporter’s knowledge was that which occurred some years later in H.M. 40th at Kwettah. The annual return gave a death-rate from pneumonia of no less than 50 in the 1,000 of strength. The disease is described as ‘pleuro-pneumonic fever.’ An idea of the character of the pleurisy is suggested by the use of the word ‘empyema.’ It was added that, within the Indus, the pulmonary complication seldom exceeded that of a bronchitis, except at the most northern stations, as at Deesa near the borders of Scinde, where pneumonia of a lower lobe in the cold season was by no means uncommon.

I have referred above to Dr. Edward Goodeve’s report on the prevalence of pneumonia at Cawnpore in 1844. Within the last seventeen years, at least, Epidemic Pneumonia has, from time to time, been reported as prevailing, in the cold weather, in Northern India. Dr. J. Kelly observed³ that, since the cold season of 1869–70, pneu-

¹ *Medical Press*, vol. i. for 1886, p. 407.

² ‘Statistical Remarks upon some of the Principal Diseases among European Troops in the Bombay and Madras Presidencies,’ *London Medical Gazette* for 1847, vol. i. p. 7.

³ *I. M. G.*, vol. vii. p. 270.

monia had been a frequent complication of the very prevalent remittent fever at Kohat, Bunnoo, and various other parts of the Punjab. In the cold season of 1870-71, Dr. Kelly observed twenty-five cases of secondary pneumonia in the first Punjab Infantry. I have spoken above of the tendency to pneumonia of the upper lobes which I observed, many years ago, in poor and ill-fed natives of Lower Bengal. Dr. Kelly found that, in six, or nearly one-fourth, of his cases, the disease was limited to the upper lobes. These cases presented many of the local and general characteristics of phthisis; but only one patient became phthisical (how far may this have been a case of intercurrent pneumonia?). Hence Dr. Kelly entitled his paper 'Phthisis simulated by Pneumonia of the upper lobes.'

Mr. Joshua Duke made¹ a valuable report on the pneumonia which prevailed in the cold season of 1874-75 among native troops marching, on the triennial relief, to the stations of the North-West Frontier of India. There were 35 admissions, with 6 deaths, all in sepoy.

What appears to have been the earliest of Dr. G. M. Giles's now famous reports, on pneumonia as observed on the Punjab Frontier, appeared at pages 235 and 272 of the 'Indian Medical Gazette' for 1883. Dr. Giles heard from Dr. Kelly, whose observations are cited above, that he was convinced that the pneumonia of the Punjab Frontier was 'a disease almost specific, and distinct from the croupous pneumonia met with in Europe.' Dr. Kelly was also strongly of opinion that the disease was, in many if not in all cases, propagated by contagion. He found, on proceeding to Dera Ismael Khan to take medical charge of the 4th Punjab Infantry, at the end of the cold season of 1882, that an epidemic outburst of pneumonia appeared to be ending. Other medical officers of the station held nearly Dr. Kelly's opinion. Dr. Giles then made examinations of the blood which are reviewed side by side with the observations of Dr. Mendelsohn in the first volume of the 'M. Times' for 1884, page 261. It is there shown that Dr. Giles gives five cases of pneumonia occurring on the Punjab Frontier. The disease appears to be broncho-pneumonia, always attended with pleurisy. The pneumonia of that locality is, in the opinion of good observers, infectious. In all Mr. Giles's cases, a peculiar micro-organism was present, both in the blood and sputa, during the whole of the illness. These organisms were similar in all the cases in which they were looked for; and, when the sputa containing these was injected into rabbits, they too were attacked by pleuro-pneumonia, to which they succumbed; and the blood, pleuritic effusions, and lungs of the rabbits were found to be crowded

¹ *I. M. G.* for July 1875; *vide* also Surgeon-Major Jessop, at p. 232 of the same vol., and Surgeon-Major Curran, on 'Croupous Pneumonia and Gangrene of the Lung,' *Ibid.* for 1877, p. 281.

with similar organisms. Dr. Mendelsohn firmly believes that the European form of pneumonia is infectious, that it depends on the presence of bacteria, and that one form of bacterium is constant in all cases. He shows that numerous cases often occur simultaneously in small districts among troops stationed at one place, in private houses and prisons (*vide* my note upon an outbreak of pneumonia which occurred, eighty or ninety years ago, in a Russian squadron at Spithead, p. 235), and that these outbreaks can often be referred to miasmatic influences, especially in connection with bad sanitary arrangements and deficient ventilation. He produces evidence to show that not only are those in immediate association with the sick in danger of taking the disease, but also that it appears capable of being carried from the sick to others by persons who do not themselves contract it. He contends that the prevalence of pneumonia is independent of changes of temperature, but that it varies with the amount of rain and the consequent rise and fall of the subsoil water. He considers that the cause of the disease is probably the *monas pulmonale* of Klebs, which Leyden and Günther obtained by puncturing the living lung. These are egg-shaped and generally occur in pairs, but also in chains of three or four. Similar, but larger, forms are found in the pleuro-pneumonia of horses. He states that the time of incubation appears to be about twenty days. He places pneumonia in that class of infectious diseases one attack of which predisposes to a second. In infected places, strangers are more liable to be affected than natives. Drunkards are peculiarly liable. Exposure to cold is a favouring, not an exciting, cause. The reviewer remarks of these two sets of independent observations, 'It is noteworthy how similarly these bacteria have been depicted by both observers,—egg-shaped bodies occurring singly or in short chains.'

More recently, Dr. Klein and Dr. Sternberg, of the U.S. Army, have been led, by separate courses of research, to consider that such of the micrococci of croupous pneumonia 'as are really infective in the sputa are accidental.' Dr. Klein 'implies that they have access to the sputa during expectoration.'¹

I should be glad to learn whether pneumonia has occurred, epidemically, in Lower Bengal in the past ten years. I am inclined to attribute the rarity of pneumonia and pleurisy in Lower Bengal in a measure to the freedom of the inhabitants from tuberculosis and strumous cachexia, to which a very large proportion of these cases are due in Europe.

For *Passive Congestion* and *Œdema*, see *Pneumonia and Bronchial Catarrh*, pp. 234-6.

I only know of one unexceptionable case of *Idiopathic* (pneumonic)

¹ *Lancet*, vol. ii, for 1885, p. 1018, in a review of the then recently published Report of the Medical Officer of the Local Government Board.

Abscess of the Lung occurring in India. I shall have to recur to it in the section on Diseases of the Liver; as it was, with apparently good reason, supposed to be a case of hepatic abscess.

Gangrene of the lung has been frequently noticed in the pneumonia of cachectic ill-fed native patients.

Pulmonary Emphysema, which, in its highest development, is especially a disease of muscular European labouring men, subject to winter cough, poorly fed, lodged, and clothed, addicted to intemperance and overworked, is not essentially an Indian disease; but traces of pulmonary emphysema are observed, particularly in cases of obstructive cardiac disease, in Indian *post-mortem* rooms. Preparations 746 to 748 in our Calcutta museum are good specimens of pulmonary emphysema in natives. In the second of these, at the lower third of the upper lobe, and on its anterior aspect, is 'an irregular opening about three quarters of an inch in diameter and an inch in depth, through which a direct communication was established with the pleural cavity.'¹

I had one noteworthy case of *Atelectasis* in the infant of an English lady. The pulmonary artery had only two healthy well-formed cusps.

In the Sanitary Commissioner's Reports for the three years ending 1882 solitary examples of this condition appear only in the two later years among the soldiers' children.²

In 1883, 8 deaths in 43 fatal cases occurred from this cause among the children born at the Madras Lying-in Hospital; and at the Ramasawmy Hospital 4 out of 31 deaths were from atelectasis.

Malformation.—Dr. Lewtas has recorded³ a case of *Congenital Hernia of the Lung* in a Hindoo, *æt.* 30. 'When he took a deep inspiration, a soft tumour was seen to develop itself just outside the left nipple.'

I treated a few miserable cases of *Phthisis Laryngea*, imported from home, but never saw an indigenous case.

Struma and Phthisis in India.

There is certainly exceeding little 'scrofula,' as expressed by the ordinary joint and glandular disease, in Lower Bengal. Allan Webb and Kenneth Mackinnon, writing at about the same time, differed in opinion with regard to the prevalence of struma in different parts of India. Mackinnon, speaking mainly from experience of localities at least 400 miles north of Calcutta, says (p. 46) that he has often seen scrofula in the form which Webb states that he has rarely met with—

¹ *Vide* case, by Dr. G. S. Sutherland, of 'Emphysema in Double Pneumonia,' in a middle-aged native, *I. M. G.* for December 1877.

² For other cases of Atelectasis, see *I. M. G.*, vol. i. No. 9, and vol. v. No. 2.

³ *I. M. G.* for August 1876.

swelling and ulceration of the glands of the neck. Webb enters (p. 125) very carefully into this question in reply to a statement made in the 'British and Foreign Medical Review' for July 1846, that 'In India, consumption is rare; scrofula rife.' He asserts that, taking Mr. Phillips's test, the existence in the glands of the children's necks of scrofulous swellings, or of the characteristic cicatrices, he had never seen such swellings or ulcerations, nor, consequently, had he occasion to open them. Dr. Webb held, for many years, the charge of two charity schools in Calcutta, with an aggregate strength of 561 children, Europeans, East Indians, and Armenians, of all ages. Of the boys, only two presented marks. The only girls having marks of scrofula belonged to one family, and were European-bred. The orphan school children were always carefully examined on admission; but, in four years, Webb never saw marks of scrofula. Here there were between four and five hundred admissions to hospital annually, 'yet not one entry for scrofula.' Dr. Webb had travelled throughout the length and breadth of India. He says, 'Among the natives of India inhabiting the lower range of the Himalayan Mountains' (a temperate region, upon which one of Mackinnon's centres of observation, Tirthoot, verges—N. C.) 'I have certainly seen scrofulous swellings and ulcers in the neck common; but in no other part of India do I know this disease to prevail generally.' So also we are told, in the Madras Report upon the medical topography of the mountainous region of Coorg, the lowest part of which is elevated not less than 3,000 feet above the sea, that there, 'phthisis is often met with, and is said usually to occur between the ages of 20 and 40.' 'Scrofula is very common, usually making its appearance at the age of puberty in the form of swellings in the neck, to which ulceration succeeds.'

Dr. Webb, and another able authority, Dr. W. A. Green, were agreed in thinking that the most common form of strumous lesion with the natives is 'bone disease of an asthenic character.' I cannot pretend to offer a valid opinion upon the question of the frequency of bone disease of tubercular origin; but it is certainly noteworthy that my Indian experience of twenty-seven-and-a-half years leaves in my memory only the solitary case of a young lady, reared in a northern temperate district, the child of a highly strumous father, who was deformed by hip disease. I never saw a case of strumous cervical glands in a native of the plains of India, a great part of which I have traversed, always keeping my eyes wide open. The result of Morehead's twelve years' experience, in the charge of the large Bombay schools, is less full than could be desired; but, as coming from so accurate an observer, it may be regarded as strongly confirming Bengal experience. He says (p. 658), 'I do not know what might be the result of the application of the test suggested by Mr. Phillips in his work on Scrofula—viz., "enlarged cervical glands discoverable by touch"; but I believe

that I am correct in saying that scrofulous disease of the joints and suppurating lymphatic or tubercular mesenteric disease are of infrequent occurrence.' These observations stand out in very strong contrast with the statistics of struma in Europe, and with one of those incisive axioms which were studded like gems throughout Thomas Addison's teaching. I was present when a fellow student, who soon afterwards died of a strumous malady, consulted him upon his case. In answer to a question, Mr.— drew himself up and said—'There is no struma in my family, sir!' 'No,' was the reply, 'certainly not—it is a remarkable fact that my family and yours stand alone in the United Kingdom as being absolutely free from all taint of struma; but, as a secret, I assure you that everybody else's family is rotten with scrofula!'

Mr. J. Murray, writing on the influence of the climate of India on children,¹ stated that we have the testimony of Sir Astley Cooper,² of Alison,³ and other experienced observers to the fact that children sent from India to England are exceedingly liable to scrofulous disease. This was probably the case under a system of therapeutics which is now discarded, but I think that the observation is not largely applicable in the present day. With thirty-seven years' knowledge of a multitude of children sent to England, I can only recall the solitary case of a boy—lodged at an atrociously miscondacted school in a London suburb, where, to my own knowledge, boys of 8 or 9 were wrapped in the rags of women's garments—who became the subject of empyemā.

In his invaluable commentary on the Diseases of Burmah,⁴ Dr. Waring observes, 'Scrofula, in any form, is very rare amongst the Burmese.'

Fully admitting the fact that nothing is more dangerous than to lay down positive rules in questions of disease, I must state confidently my belief that, *if an individual lands in Lower Bengal, with perfectly healthy lungs, and lives well, prudently, and carefully, without extraordinary exposure or more than usual mental or bodily fatigue, he will, residing there, never be attacked with phthisis, although he may be a member of a phthisical family.* Against this, I may cite Joseph Ewart's observation, that people who would probably have become phthisical at home are very liable to dysentery in India; and also the fact, noticed by several authorities, that strumous subjects are especially liable to hepatic abscess in India.

The opinion given in italics above is the outcome of my personal experience of non-military Europeans throughout my Indian service, and of much inquiry. More than thirty years ago, I heard of one case; but as, there, the patient had been cooped up in the fore-castle of a small

¹ *I. M. J.*, vol. ix., O. S., p. 525.

² *Lancet*, vol. iv. p. 400.

³ *Outlines of Physiology and Pathology*, p. 471.

⁴ *I. A.*, No. 1, p. 101.

vessel, my late honoured friend, Dr. Henry MacCormac, would doubtless have considered that the tuberculosis was artificially produced. As the effect of removing a case of phthisis from Europe to Calcutta is, in the large majority of cases, nearly that of burying a dead body in a hot-bed, there is a great deal of pulmonary consumption, especially among sailors, in the European wards of the Calcutta hospitals. So also, in the cold season, many cases occur among natives ; but, in a rough way, this fact is very significant—The London hospitals formerly used, as rigidly as possible, to exclude cases of phthisis, as I believe they still do, seeing that special aid is now afforded to these cases ; still, during the many years in which I frequented it, the *post-mortem* room at Guy's was rarely without a case of pulmonary tuberculosis. In Calcutta, it was a standing rule that all phthisical applicants should be admitted to my wards, which, although sometimes nearly filled with cases of consumption, were never overcrowded. Seeing that my own share of beds for native men was only sixteen, this must be regarded as representing no great prevalence of the disease in a city numbering about half-a-million of inhabitants, and in its thickly populated vicinity.

To me, the arguments contained in Morehead's chapter xxiii. do not appear to bear so directly as could be wished upon the very important question—*to what extent does phthisis ORIGINATE in India ?*

His remark, immediately following the heading *Causes*, that 'the erroneous opinion at one time entertained of the rarity of phthisis pulmonalis in tropical countries has been long since corrected by the medical statistical reports of the British army, and information from other sources,' would be a direct and accurate reply to the mischievous idea, formerly entertained at home, that the climate of India is favourable to phthisical cases ; but it must be apparent that the prevalence or rarity of phthisis in our European force in India in no way illustrates the *causation* of phthisis in that country, seeing that, in all probability, a very large proportion of the men who succumb to phthisis in India were the subjects of tuberculosis before they reached that country. I feel little doubt that the records in the Director-General's Office would supply materials for a return showing the number of men first attacked with phthisis after five years' service in India. This would go directly to the point at issue. Morehead says, 'If the history of these' [Byculla] 'children be traced after they have grown up and left the schools, it will be found that phthisis pulmonalis is a cause of death sufficiently common. I can bring to my recollection several cases in proof of this.' He shows that the number of natives suffering from phthisis admitted to the J. J. Hospital, Bombay, during six years, was 445, not many out of so large a population, upwards of 566,000, as that city then had.

We are told, in the 'Medico-Topographical Report,' p. 47, that, in

Madras, phthisis 'has been frequently met with in Indo-Britons,' and that it 'has also been seen in European subjects about the age of puberty, and within the first two or three years after arriving in India, but is of rare occurrence after that period; and, in natives, true tubercular phthisis has comparatively been seldom met with.

My friends, Dr. T. W. Wilson¹ and Dr. Joseph Ewart,² offer us, in monographs which are replete with facts of the greatest importance to Indian practitioners, a large view of this class of diseases as they have prevailed of late in India. Dr. Wilson shows that, formerly, a great deal of tuberculosis and phthisis was overlooked, especially in native jails; and that cases of phthisis with colliquative diarrhœa were frequently registered under the heads of 'Diarrhœa' and 'Dysentery.' At the Medical College Hospital, I always bore this observation in view, and it repeatedly occurred to me to find that an emaciated native, who complained of nothing except an alvine flux, was, in reality, dying of pulmonary consumption. Dr. Ewart shows that, in the decennium 1857-67, 454 cases of phthisis occurred in natives treated in the Calcutta M.C. Hospital in a population of about half a million.

[We have seen above that 445 cases of phthisis in natives were treated in the less capacious Jamsetjee Jeejeebhoy Hospital, Bombay, in six years.]

Ewart shows that, in the six years 1871-76, the ratio per 1,000 of strength treated for phthisis amounted to 9·0 among the soldiers of the European army in India. In the native army of the Bengal Presidency, the corresponding ratio amounted to 2·5. In the jail population, it varied from 1·5 in the Central Provinces to 8·9 in that of the Gangetic Provinces and Oude. In the aggregate of the jails of the Bengal Presidency, the ratio was 4·2 per mille. He further observes that, according to Aitken, the ratio of admission for phthisis in the British army, from 1861 to 1862, ranged from 9 to 10 per 1,000; and in the American army (1862-63), from 8 to 10 per 1,000. Consequently the prisoners in the jails of the Gangetic Provinces and the soldiers of our European army throughout India were then scarcely less prone to phthisis than soldiers at home were. He draws the same deduction with regard to the women and children in European barracks.

In happening to take the statistics of the six years 1871-76, *i.e.*, in showing that the admissions for phthisis in our European army in India were 9·0 per mille, Dr. Ewart places the matter in its worst aspect. Thus Mr. Macpherson found that, in the four years ending 1853-54, in the Bengal Presidency, 'pulmonary consumption brought under treatment $\frac{2}{3}$ per cent. of strength, less than one-half the number

¹ 'On Tubercular Disease in the East,' *I. A.*, No. 3, p. 182.

² 'On Scrofula, Tuberculosis, and Phthisis in India,' *Transactions of the Epidemiological Society*, vol. iv. part iv. p. 597.

in England, and of deaths to treated $44\frac{9}{10}$, or 30 per cent. less.' 'Spitting of blood contributed to the admissions $\frac{1}{7}$ per cent. of strength, or one-third fewer than in England; and of deaths to treated $\frac{7}{10}$ or $1\frac{1}{2}$ per cent. less.

The Returns for the whole European Force in 1881 give an admission rate of 7·1. In the native army it was 2·9.

The Returns for 1881 show that only one *child* of a European regiment was admitted (in Bombay) for 'scrofula' in the army of India, the strength being 6,548. There were, during the same term, 43 admissions of children for phthisis, and 24 deaths. The admissions for tabes mesenterica were 15, with 1 death. Among the *women*, there was one case of scrofula, which was fatal; among them, the admissions for phthisis and hæmoptysis were at the rate of 11·2 per mille of strength.

I think that, undoubtedly, the great prevalence of phthisis in European barracks in India points to the necessity for an inquiry as to whether these buildings, or, at least, those of them in which a large amount of phthisical disease occurs, are properly ventilated and are not overcrowded. Then a plain suggestion offers itself. A party, 1,000 strong, is drawn up on a wharf just before embarkation for India. We know that, healthy as their appearance is, some nine or ten of these men will be admitted to hospital for phthisis within the next twelvemonth. Ought not these, and more, to be eliminated from the draft, seeing that, in all probability, all in whom the disease is latent will die in India within their first year's service, and would, almost certainly, wear on much longer at home?

The most extreme case of military tuberculosis I ever saw occurred in a native prisoner in the mud jail at Purulia. Wherever I was able to trace fully the history of a native phthisis case, I found that its subject had inhabited an ill-ventilated brick or mud building. I never succeeded in tracing a case to a bamboo hut. Upon Dr. MacCormac's doctrine, I believe that the immunity from phthisis of well-to-do Europeans in Lower Bengal is mainly due to the free ventilation of their sleeping-rooms. I regard this very remarkable immunity as a strong argument against the heredity of phthisis.

I think that I may say confidently that none of the many phthisical European sailors whom I treated lived, after their arrival in India, so long as five months. I cannot recall a single case of 'Discharged Relieved,' although there were many of these in the native wards. The picture of a European dying of phthisis in Calcutta is one of the most painful in hospital experience. The bowel complication usually takes on a dysenteric character and occasions extreme distress. The heat of the climate induces and greatly aggravates colliquative sweating. Every one who thinks of sending a phthisical patient to a hot climate should look at Severn's masterly sketch of the face of Keats in

his death sweat at Rome. In India, I never saw the disease borne with that cheerful hopefulness which characterises many of these cases at home. The phthisical European in India is generally so despondent, irritable, and captious that I used often to approach the beds with an uneasy doubt—what new grievance is to be adjusted to-day? Hectic is usually very pronounced. If these unfortunates arrive in the cold weather, the next hot season will be fatal; if in the hot weather, and they survive it, the change of temperature at the beginning of the cold season will probably excite intercurrent pneumonia, and kill them. In nearly all European cases, the destructive process is exceedingly rapid. A young gentleman, who had suffered from hæmoptysis in Scotland, came to my house soon after landing. He appeared to be in fair health. Shortly afterwards, I attended him in an attack of hæmoptysis from the left apex. The hæmorrhage readily passed off, and there remained no evidence of lung disease anywhere, save a little increase of sub-clavicular vocal resonance (which is frequent in healthy persons) on the affected side. I did not see him afterwards. A week or two later, an accomplished brother officer, having carefully examined his lungs, remarked that, but for the history, he should have considered the chest perfectly healthy. From this time ‘decline’ was rapid, and he died in about three months. His lungs were brought to me. Tubercle had proliferated to so enormous an extent that the organs were solid. He must have died suffocated. There was no history of intercurrent pneumonia.

Morehead was inclined to subscribe to the opinion that ‘phthisis runs a more rapid course in warm than in cold climates,’ on the ground that ‘in all diseases which include destructive degeneration of structure and co-existing hectic fever, the rapidity of the course will bear relation to the number and degree of the debilitating influences to which the individual is exposed.’ Among such influences, he enumerates heat, malaria, a greater proclivity to intestinal ulceration; and, as causes of intercurrent pneumonia and bronchitis in natives, weak constitution, insufficient clothing and lodging.

At p. 128* of his great work, Webb draws a most striking picture of the misery caused by the fatal impression, then prevalent, and not yet quite extinct, in England, that the climate of India is favourable to the strumous and consumptive. He says, ‘I have seen medical men, clergymen, officers in the service, &c., who have told me, when surprised with the fatal turn of their cases, that they expected to *get well here*. I have seen young and beautiful European ladies carried off with appalling celerity. I have seen quite young soldiers who, if they do not quickly die, are sent home wholly unfit for this or for any other service.’

The question, ‘*Is the climate of Indian Hill Sanitaria beneficial in scrofula, tuberculosis, and phthisis?*’ has been most thoroughly

argued out by Dr. Joseph Ewart.¹ I believe that all medical men of Indian experience will agree in the validity of his conclusions, (1) that the hill sanatoria of India are beneficial to persons afflicted with scrofula only; (2) that they are useful, in the summer heat, (a) in uncomplicated and simple tubercular consolidation, (b) in tubercular affection in which previous inflammation of the adjacent lung and the bronchial mucous membrane has subsided, and (c) where this is proceeding in the direction of repair by absorption, cicatrisation, or cretification; and (3) that they are injurious, probably at all seasons, but in an aggravated degree in the cold season and rains, to all cases of active and advancing pulmonary consumption.'

I have the following very scanty evidence tending to show that the climate of the East and West Indies is not *certainly* fatal to European consumptives. A London surgeon, who was phthisical, spent some months in Jamaica. He appeared improved in health on his return; and, subsequently, his case proved to be remarkably chronic. A strong-looking middle-aged gentleman told me, in Calcutta, that, having, when young, suffered very severely from hæmoptysis, that excellent man and physician, Thomas Hodgkin, sent him to Jamaica, where he recovered. A young medical officer, who arrived in Calcutta *circa* 1858, was attacked with hæmoptysis almost immediately upon landing. When he was convalescent, the head of our department, Sir John Forsyth, whose secretary I was, said to me, 'What can we do with W——? We can neither send him home nor to the Upper Provinces. He shall go with that detachment to Dibroghur in Upper Assam. He will have a pleasant voyage of some days up the Braham-pootra, and the climate is temperate.' Dr. W—— had no return of his disease. Most of his service was at the above station, and he has lately retired with the appearance of a remarkably powerful and healthy man. I have seen Dibroghur, and certainly do not consider that the stream of consumptive patients ought to be directed thither; but there, and in the hills of Jamaica, there is a moderate climate which may probably agree with some consumptives. These three exceptional cases do not appear to bear upon the rule, denouncing hot resorts in phthisis, laid down above. I had some experience of recovery from incipient phthisis in natives of India. I need hardly say that hard-working students often suffer from transient hæmoptysis. Sir Astley Cooper did, and directed that those who examined his body should look for the traces. I had very slight hæmoptysis, and long afterwards coughed up an earthy concretion. Several of my military class students, Mahomedans, had severe attacks of hæmoptysis from which they so completely recovered that when, upon the completion of their three years' term of study, it was my duty to examine them, I did so

¹ *Transactions of the Epidemiological Society*, vol. i. for 1881-82, p. 26.

with especial care, and could not find any evidence of pulmonary lesion. In examining the body of a dacoit (professional robber) who had been hung, I found a cavity, about as large as an orange, in the upper lobe (below the apex) of the right lung. It was neither a dilated bronchus nor a pneumonic abscess, but clearly a phthisical vomica in process of recovery. There were no tubercles. He was of middle age, gaunt, but wiry, and his history showed that his habits were only too active.

I lately heard of the very sad, but interesting, case of an officer who, before he went to India, was considered to be so much predisposed to tuberculosis that he was strongly advised not to marry. After some years' Indian service, he became and continued to be a very strong and healthy man. He married; two of his children, having arrived at adult age, have died of phthisis, and three others are threatened.

In recent conversations with my friends, Brigade-Surgeons J. Barclay Scriven and Dr. William Palmer, who are equally experienced in the diseases of the North-Western Provinces and in those of Lower Bengal, I learnt that, although they fully agree in considering that the climate of the latter is most inimical to sufferers from phthisis, they had cases in which the dry atmosphere of the plains of Upper India, say Lahore and Ghazeepore, stations of which they respectively long held medical charge, appeared to be decidedly beneficial. Further observations on this point are very desirable. I saw and carefully examined, not long before his death, a military officer who, I was informed on good authority, had suffered, in Upper India, from unmistakable phthisis for ten years.

Treatment of Phthisis.

I have always held to and acted upon the conviction, expressed in one of my earliest publications,¹ that bronchitis is not essential to and is separable from phthisis, and that, consequently, the leading indication of treatment in this disease is *to free the lungs from bronchitis*, and to keep them free. Cough does not, of necessity, attend phthisis. Several of my native phthisical patients came into hospital without cough. Edward Goodeve made an excellent observation upon this point in his Cawnpore Dispensary Report for 1845. 'We make it a rule to examine the condition of the chest in all cases of chronic diarrhœa, dysentery, and so-called remittent, but frequently hectic, fever, and in continued fevers in the cold season. The patients often make no complaints of chest disease themselves, their attention being concentrated on the symptomatic fevers and diarrhœas, or failure of

¹ 'Observations on the Treatment of Phthisis,' *London Medical Gazette*, vol. ii, for 1840, p. 782,

strength. It is necessary to question them closely, to watch the general symptoms of lung affections, and generally to resort to auscultation before coming to a conclusion as to their real complaints. Within twelve months, we have met with every form of pulmonary disease, except the malignant ones.' . . . 'Tubercular phthisis we have had an abundance of.'

This absence of bronchial complication in phthisis has also been observed in England ; but, here, bronchitis is so common that phthisical lungs rarely escape it. A *Hem* is all that is requisite for the expectoration of blood and pus from vomicae. If I had a phthisis ward, I would write above the entrance COUGHING IS NOT PERMITTED HERE. In every case that I have treated, this indication has been constantly borne in view. I cannot boast of marvellous success, but I have had many favourable cases, especially in natives, whom I have treated from the first attack of hæmoptysis onward. I relied greatly upon extensive counter-irritation, long maintained. As a general rule, neither Europeans nor natives, in Calcutta, could digest cod-liver oil in phthisis.

In India, the condition induced by tubercular ulceration of the intestines in phthisis is, in many cases, not diarrhoea, but dysentery, and the suffering from this cause is often very great. In reports of the recent case of King Alphonso of Spain, the intestinal complication of phthisis, which caused death unexpectedly, is described as 'dysentery.' This is one of the forms of dysentery in which the ipecacuanha treatment is inadmissible. My chief reliance was upon opium (a valid anti-dysenteric) and demulcent food. In chronic cases, I have sometimes staved off bowel irritation by dealing very carefully with the troublesome constipation which is its usual precursor. The patients are often most urgent for purgatives, but I never allowed more than the very smallest doses of castor oil. In the last case that I treated, there was constipation for a year before the patient's death, and the bowels were never allowed to become loose. In several miserable cases of excessive ulceration of the rectum, some relief was obtained by small enemata of pure opium rubbed up in mucilage of acacia, and the application of warm anodyne poultices externally.

I repeatedly noticed that, on a chilly gusty morning in the rains, two or three patients would be attacked with *Hæmoptysis*. This was always commanded by ergot.

I had several cases of phthisis in which *Pneumo-thorax* occurred. Mine were ordinary typical cases, except that of a middle-aged native who was walking about the ward with no signs of thoracic distress. I was surprised when my house physician called my attention to the splash and the metallic tinkling.

I saw a case of *Primary Medullary Cancer of Lung* many years ago in London. I did not meet with this condition in India, and I do

not find notice of any specimen in Ewart's Catalogue. Assistant-Surgeon Dr. Jayakar has recorded¹ a case (encephaloid or melanotic) occurring in a native aged 35 years. I have already cited, under the heading of Cancer, the case of a native, related by Allan Webb at p. 1 of his 'Pathologica Indica,' in whom there was melanosis of the lungs and heart.

Pleurisy.

Placing aside cases in which pleuritic inflammation was caused by wound or occurred intercurrently in phthisis, acute rheumatism, or Bright's disease, or where liver abscess made its way into the chest, I only recollect to have seen one case of acute idiopathic uncomplicated pleurisy in Lower Bengal. This occurred in a European who did well. I repeat that my observation applies only to the Delta of the Ganges.

Mackinnon says, 'I have seen pleuritis prevail in an epidemic form'; but I can only find one other notice of this disease in his works. 'Pleuritis I have seen prevailing among prisoners in jails at certain seasons.' In the chapter on Pneumonia, I have mentioned that Dr. W. A. Green recorded [Webb's 'Pathologia Indica,' p. 106*] a remarkable prevalence of *Pleuro-Pneumonia* among the prisoners in the Midnapore jail in 1844 and 1845, and that pleuro-pneumonia occurs on an extensive scale in Upper India.

In one of his latest publications, Mackinnon barely repeated the fact² that he had '*seen pleuritis prevail in an epidemic form.*' A few details would have been most valuable. This probably occurred in his up-country experience.

The S. C. R. for 1880, in which year we have seen that pneumonia was remarkably prevalent, give 599 admissions for pleurisy with 61 deaths in the native army. In 1881, there were 377 admissions with 17 deaths. In 1882, there were 266 admissions with 14 fatal cases. The average strength of sepoys, during these three years, was 118,630. In these three years, the deaths, from this cause, of European soldiers, with an average strength of 58,571, were five, two, and four.

Macpherson's tables give 688 cases of pleurisy, and 33 deaths, as occurring during the 8 years ending 1853-54, among the European soldiers in the Bengal Presidency. The percentage of sick was $\frac{2}{3}$ —twice as large as in England—and the percentage of deaths to treated $4\frac{1}{5}$, one-fourth larger than at home.

Pleurodynia is very common in Lower Bengal, and is often so acutely painful as, at first sight, to resemble pleurisy. My European male ward was so ill-placed that the beds stood in a thorough draught. One damp windy morning, I found the three patients

¹ *I. M. G.*, vol. v. p. 79.

² *I. A.*, No 5, p. 128.

nearest to an open window complaining of pleurodynia on the windward side.

I never saw a case of *Idiopathic Empyema* in a native of Lower Bengal. Prep. 608 in the Calcutta Museum, however, represents 'displacement of the heart of a native of Bengal from empyema.' A very few other cases are on record. One was reported at the Calcutta Medical Society in 1880; and my friend, Dr. W. Burns Beatson, now of Bath, described ¹ one occurring in a native of Azimghur, who contracted the disease at Dacca, where he had been for some time resident. In 1882, only 4 cases, one of which was fatal, occurred in the native army throughout India—all in the Bengal Presidency—most probably up-country. There can be little doubt that the rarity of pleurisy and of this condition is due to the great immunity of the natives of the plains of India from strumous disease.

I knew two gentlemen, a member of the Civil Service and a tea planter, in whom there was great falling in of the thorax consequent upon empyema in youth. In the former, the deformity and the deficiency of lung expansion were more extreme than I have seen in any other case, yet he was a noted cricketer. Both enjoyed good health in India, but were accidentally killed early in life.

I had various cases of *Hydrothorax* arising from ordinary causes. In one of these, my diagnosis cannot have gained for me any credit with my class. A northern sailor, from whom I could obtain very little information upon the history of his case, was brought to my ward in considerable dyspnoeal suffering. He was a muscular square-built man, over 30 years of age, his face and neck were much congested, and the veins of the neck extremely full. Great bulging of the right thorax. Absolute dulness extending over the right pulmonary and hepatic and the cardiac regions. Absence of respiratory and vocal sounds on the right. The edge of the liver was felt below the ribs. A soft systolic aortic bruit extending upwards. I considered this to be aneurysm of the ascending aorta, filling the right thorax, pressing the collapsed lung upwards and the congested liver downwards. This was all wrong. When he sank it was found that the right pleural cavity was distended with clear serous fluid, apparently an inflammatory product, as the serous surfaces bore traces of pleurisy. It was evident that the bruit was caused by outward pressure on the artery. Few of my cases have caused me so much after-thought as this has done; but, if I had another like it, I am not at all assured that I could judge it better. I have seen enormous aneurysms of the ascending aorta, none certainly as large as this would have been; still the size of aortic aneurysms can hardly be said to have a limit.

C.M. Prep. 780. 'Extraordinary lenticular vegetations projecting

¹ *I. M. G.*, vol. i. p. 60.

from the costal pleura like small buttons. Some of these are globular, others more or less elongated. They arise by a broad base, and are covered by a smooth membrane continuous with the pleura, which is greatly thickened. They vary in size from a millet-seed (or small point) to those of a kidney-bean. From a native of Bombay, who died of dysentery.'

I saw cases, in Europeans, of *Abscess* and of *Non-Malignant Tumour* of the *Anterior Mediastinum*.

A very noteworthy case, in which a *bullet* had remained for 8 days in the *anterior mediastinum* of a Hindu, *ætat.* 40, is given in the Madras Civil Hospitals' Report for 1883, p. 73. He had no symptoms of any kind beyond some sanious discharge and inability to assume dorsal decubitus without aggravation of the otherwise slight pain. The wound was in the third left intercostal space near the sternum. On removing a cubical piece of intercostal cartilage with the bone pliers, a bullet was discovered to be lying free upon the pericardium. It could be rolled about in the anterior mediastinum in all directions, and the pulsations of the heart were easily felt with the finger. The bullet was removed with dressing forceps. It was globular and weighed six drachms. The wound was healing steadily, and it was expected that the man would soon be well enough to be discharged.

DISEASES OF THE DIGESTIVE SYSTEM.

Diseases of the Mouth.

I have seen many Europeans and East Indians born in India with *Hare-Lip*, but I never saw that condition in a native.

Natives are liable to *Epithelioma of the Lower Lip*.

Stomatitis.—A moderate amount of stomatitis frequently attends idiopathic sloughing of the cornea in ill-fed Bengalis.

Aphthous Stomatitis, *vide* p. 120, is very common in European and native children, especially those who have suffered from diarrhœa in teething. Where the soft palate is affected, the case frequently has much the appearance of diphtheria; and, both in children and in adults, I have repeatedly considered that I had grounds for thinking that this affection has been mistaken for diphtheria—a serious error when the feelings of the patient's friends and of the patient (when a delicate nervous lady, with health broken by long residence and frequent sickness in India) are considered. The subjects of *Morbus Bengalensis*, *Diarrhœa alba*, and *Sub-acute Dysentery*, frequently have aphthous mouths, and I suspect that few old Indians are entirely free, as long as they live, from more or less of this annoyance. Good non-malarial air, quinine, and a generous diet, with the local use of port wine, used as a wash, and mel boracis are the chief indications of treatment. The

best application is a saturated solution of alum; the most painful aphthous spot heals after having been twice touched; but, as alum injures the teeth, it should be applied with a camel's-hair brush.

In 1878, Mr. Raddock reported¹ that, in the preceding May, stomatitis attacked the sepoy of the 31st N. I. on the voyage to Malta; 30 cases, or 4·14 per cent. of strength, suffered. There were constipation, foul tongue, small, red, and very painful pimples [aphthæ?] on the edges of the tongue, malaise and anorexia. In worse cases, there were pimples all over the tongue, and patches of abrasion of epithelium, and inability to eat from pain in masticating. In the severest cases, there were superadded fever, pain in swallowing, sore-throat, hoarseness, loss of voice, cough, and salivation, and the mucous membrane of the throat was much congested. There was no appearance of scurvy, the affection occurred principally among the beef feeders. Mr. Raddock believed the disorder to be one of the digestive system, and the occurrence of urticaria in several of the cases strengthened this view. Nitro-muriatic acid internally, carbolie acid gargle and rice diet were employed. The force of the attack was broken in four or five days. The R. wing and head quarters of the regiment had not any case on board their transport, but they were attacked four days after landing at Malta, on June 3, and when the whole regiment was together again; and, from that time, all the companies, and all classes and castes of men, were alike affected, and to a large extent. When the report was closed, on June 22, it was said, 'The disease is now on the decline, and seems to have given way to dysentery.'

Thrush occurs in sickly Indian children, and, in them, as in aged persons dying in that country from various chronic diseases, is generally a portent of speedy dissolution.

Cancrum oris has been dealt with, as an expression of paludal cachexia. See page 257.

In 1881, the Returns show the death of one European soldier in India from *cancrum oris*. His is probably the case reported² by Mr. P. W. O'Gorman. There were enlarged spleen and pancreatic disease.

A healthy middle-aged man long put up with great swelling of the cheek, doubtless seated in the *obstructed duct of a buccal gland*, which burst inwards, giving exit to several ounces of honey-like fluid. A barrister had a very considerable, but smaller, swelling in the same locality. It suppurated and pointed outwardly. Dr. R. C. Saunders has recorded³ a case of *Salivary Fistula*, consequent upon an abscess of the cheek in a Mussulman. It was cured by seton.

Ramula.—One of the first specimens contributed to the Museum of the Calcutta Medical Society was a '*calculus taken from the sublingual duct*,' by Dr. John Adam. In 1836, Mr. Charles Mackinnon

¹ *I. M. G.* for September of that year.

² *Ibid.* for 1852, p. 15.

³ *Ibid.*, April 1875.

forwarded to the same collection 'a salivary calculus, which had been extracted from below the tongue of a native.' It was as large as a filbert, and weighed upwards of half an ounce.

Assistant-Surgeon Cheytun Shah removed, apparently from the duct of the sub-lingual gland, a grain of barley covered with concretion. The lad, a native, came complaining that, about three years previously, the grain had stuck in his mouth and disappeared below the surface.

Dr. Wertheimer has lately recorded¹ a case in which a gooseberry seed was found obstructing Warton's canal.

Preps. 64 and 65, S. 18th, in Sibthorpe's Catalogue are 'Concretions removed from the sub-lingual duct of a gouty subject'; and 'salivary concretion from duct of the sub-maxillary gland.'

In 1835, the case of a Hindu boy of nine, a patient of Mr. Malcolmson's, was discussed at the Calcutta Medical and Physical Society. What appears to have been a *Ranula* formed a swelling below the jaw extending from ear to ear, over the angle of the jaw, and below the head of the sternum. A tentative puncture gave exit to 20 oz. of a glairy brownish liquid. A hard substance remained, and proved to be a portion of the fluid inspissated. The fluid again collected, and, a tent failing to cause contraction of the sac, this was opened below. The interior exhibited an extraordinary appearance. 'From behind the ears, over the angles of the jaw, and down to the chest, it seemed as if carefully dissected, the blue veins and parotid glands shining through the cellular membrane.' A small gland was removed, and the lower maxillary artery was divided. An abscess formed which burst into the mouth, but he was well in a month. Mr. Egerton said that he had never seen a case of ranula in India. Dr. Spens replied that he had then under his care a case in a native.²

My friend, Dr. Herbert Baillie, recommends that the tumour be seized and secured by a pointed hook and, a large portion of the cyst being snipped out with curved scissors, the sac be filled with tincture of iodine, B.P., which, after remaining a few minutes, is allowed to escape by the mouth. Its place is supplied with oiled lint, which is renewed daily after the cavity has been well washed out. There is temporary swelling, but rarely much suppuration.³

Epithelioma of the cheek.—One of my first patients in India was a prisoner in the jail of Purulia, Maunbhoom, with an epithelioma which had perforated his cheek, midway between the ear and the commissure of the lips, so completely that the little finger might have passed through. I removed it, and several enlarged glands extending up under the malar bone, by two semicircular incisions. The wound, brought together by interrupted sutures, did well, and there was no

¹ *Bulletin Méd. du Nord*, December 1885, and *Prov. Med. Jour.*, April 1866.

² *C. M. T.*, vol. viii., part i., p. 13.

³ *I. M. G.*, vol. v., p. 36.

salivary fistula ; but he died from malignant disease of the sub-maxillary gland.

Sir Joseph Fayrer has narrated cases showing that hideous destruction of cheek, gums, and alveoli is produced by *Salivation* resulting from the misuse of mercurial fumigation by native practitioners.¹ See also *antea*, p. 330.

Dr. Cayley has reported² a case in which a Hindu had the structures of his mouth so agglutinated by adhesions, consequent upon salivation, that he could not separate his jaws, and was only able to get a little nourishment by rubbing soft rice through his closed teeth. Consequently, he was almost starved. An operation was successful.

Mr. Holmes has described and figured³ a remarkable *Exostosis*, which formed in the *Antrum* of a native, a patient of my friend Dr. Duka. On removing the front wall of the upper jaw, the large mass of ivory-like bone was found lying quite loose in the cavity, its attachment having been separated either by its own weight or by some accidental violence.⁴

The various kinds of *Tumour* to which the *Jaws* are liable in Europe are of rather frequent occurrence in India. As these growths are frequently allowed to proceed far before relief is sought, operations for the removal of the jaws are among the chief triumphs of surgery in India. Richard O'Shaughnessy was one of the first who undertook these cases, and he published a valuable work on the subject. Webb and Partridge had many successful cases.

Sir Joseph Fayrer has devoted a chapter in his 'Observations' to the history of twenty-four cases, all natives, of non-malignant diseases of the jaws which came under his own treatment.

Dr. Maclean, of Netley, reported a case of *Epulis* in the 'Hyderabad Proceedings.' The five specimens, numbered from 367 to 371, in Ewart's 'Catalogue,' represent this disease, in tumours of various sizes, arising from the gum, or more or less involving the alveoli, jaws, or cheeks. I successfully removed an epulis, of the size and shape of a lime, from the front of the lower jaw of a native woman at Chittagong.

¹ Commenting upon this want of discrimination in employing mercury, Kenneth Mackinnon wrote : ' This fact need not excite our wonder, when we bear in mind that, thirty years ago, English practitioners administered it, according to the quantity of saliva discharged, the teeth and sometimes parts of the jaw-bones falling into the spittoon.' I have seen men, in a naval hospital, sitting up all day in bed, not spitting but draining from the mouth into a tin utensil. A few of these cases filled a spacious ward with an abominable characteristic odour. It was a general, although probably erroneous, idea that, in this close (because the patients must not be chilled) and fetid atmosphere, salivation occurred earlier and more severely than in wholesome air.

² *I. M. G.*, vol. vii., p. 184.

³ *Treatise on Surgery*, third edition, p. 433.

⁴ The case is narrated in *Path. Trans.*, vol. xvii., p. 256.

It appeared to be still confined to the gum. Apprehending the sharp arterial hæmorrhage which occurred, I had a pointed actual cautery ready. This was quite effectual, and almost painless.

Diseases of the Teeth occur in India nearly as in England. Like all colonials, the children of European parents, born in India, are very liable to lose their permanent teeth early. Care should be taken in giving strong acids, especially dilute sulphuric acid in quinine mixtures. Seven children of an officer, born in India, had great trouble with their permanent teeth; the eighth, who was born in England, and was only taken out for a few months as a child to India, where his health was good, had very sound teeth.

When at Howrah, I saw, in a native infant, one of those cases in which it is said that the *child is born with teeth*. In the place of the incisors, the gum was capped with a horn-like growth, which was hard but appeared to be no thicker than the child's nails.

Dr. Cullen mentions the case of a child, said to be only two months old, which had cut the two lower incisors. A fortnight later, an upper incisor was cut.¹

European children in India, for the most part, cut their teeth earlier than the infants of ill-fed natives and of most East Indians do. In the children of the latter, it often becomes a very hard battle for life when the time for the appearance of the canines comes before most of the teeth which ought to have preceded them have been cut. I always dreaded these cases. I, for some years, saw a great deal of European and East-Indian children during their first dentition. I do not recollect a single case in which the usual order in which the teeth are cut was at all broken. I could always judge correctly which teeth were coming next. A writer in the 'Indian Journal of Medical Science'² reported the cases of two children, in one of which the four canine teeth, and in the other four molars, made their appearance, neither infant having previously cut any teeth. Both children were taken to him in a dangerous state, the former having severe febrile symptoms and diarrhœa of some days' duration, the other in convulsions. Their respective ages were eleven and eight months. The front gums, where any of the eight incisors might have been expected to be the cause of suffering, were perfectly soft and cool; but great heat, redness, and swelling existed, in one case, in the situation of the canine, and, in the other, of the molars. The free use of the gum lancet, calomel and castor oil, were followed by complete relief.

I recollect to have had to pay seven visits a day to a European child with *narrow bird-shaped jaws*, in which the teeth, as they came up, had not sufficient room. The father, although a remarkably good-looking well-made man, had similarly malformed jaws which greatly affected his speech. Nothing can be more futile, practically, than

¹ *I. M. G.*, November 1876.

² Vol. i. p. 244.

the contention that, as lancing the gums injures the enamel of the teeth, we should refrain from doing so as much as possible—seeing that health, probably life, is at stake, and that moderate injury to the enamel of the milk teeth is a minor evil. I never tried to injure the enamel, but every stroke of my gum lancet went completely across the tooth, touching it, and just beyond it, in two incisions, Saint Andrew's cross-like. Even in the most pallid children, in highly malarious localities, I had no fear of troublesome hæmorrhage and never saw it; indeed, I was always glad to see rather free bleeding. I have, however, spoken of exceptional cases in which very excessive hæmorrhage occurred, in the chapters on Malarial Cachexia and Purpura Hæmorrhagica. Mr. C. W. Fuller¹ attended a European child, six months old, in which repeated attacks of formidable hæmorrhage occurred from the gums over the upper and lower incisors which he had lanced after the child 'had been taking a small quantity of calomel' for fever. The bleeding was stopped by the application of the actual cautery to the bleeding spots.

The natives clean their teeth daily. The practice of chewing *pan* (catechu, betel-nut, and cardamoms) appears to agree well with the teeth and gums, except when, as is often the case, an addition of lime is made. Unless great care be taken, this forms an incrustation between tooth and gum, and tends, ultimately, to lift the tooth out of its socket.

Salivation and scorbutus have been fertile sources of disease of alveoli and loss of teeth among Europeans in India. As there are no professed dentists in that country, out of the Presidency towns, every Indian surgeon ought to be a good operative dentist.

Tooth-drawing, like midwifery practice and the excision of non-malignant tumours, obtains for us, in India, fully as much professional reputation as the nature of the work accomplished merits. At Chittagong, our judge, who I am certain believed that he knew far more of my profession than I did, came with his brother, the magistrate, to my dispensary with tooth-ache. I took out the peccant molar with that abominable instrument the tooth-key. Not being a strong man, he nearly fainted. After that, he generally deferred to me in surgical questions, while he never ceased to maintain his own supremacy as a homœopath.

I received a severe lesson in dentistry on my way out to India in medical charge of troops. An Irish recruit complained of tooth-ache, and pointed to the last molar. I drew it, thinking that it was decayed behind, but found that it was perfectly sound. He then pointed to the next tooth which, when drawn, proved to be equally perfect. I then talked to the man and found that he was a 'softy' of very low type, who ought not to have been enlisted.

¹ *I. M. J.*, vol. i., p. 288.

While officiating as Inspector-General of Jails, I had my attention drawn to the case of an elderly English gentleman who, having been convicted of breach of trust, was undergoing imprisonment in the Calcutta jail. It appeared that his teeth were in such a bad condition that he had quite ceased to be able to masticate, and his friends begged that a dentist might be allowed to make him a set of teeth at their cost. When this was done, he appeared to enter upon a new lease of life. At about the age of eighty, an honoured brother officer, who had been salivated in India nearly forty years previously, became half starved and very seriously debilitated from the same cause. No old Indian who wishes to live long ought to avoid the dentist's aid.

But this should be employed with discretion. My friend was a very strongly built man, and I believe that the wrenching away of a molar with its alveolus gave almost as severe a shock to his nervous system as the amputation of a hand would have done. A weak constitutioned, free living, and diseased Londoner, *ætat.* 62, shocked me by telling me that he had submitted, at one sitting, to the tearing out of the whole of his remaining decayed teeth and stumps. I consider it probable that his death, which occurred a few weeks afterwards, from a head affection allied to D. T., very similar to many others from which he had recovered for some fourteen years, was indirectly due to the shock which his faulty nervous system must have then sustained.

In India and at home, I never saw *Glossitis* except as a result of mercurial salivation; but, in India, remarkable cases of what has been called '*Chronic Hypertrophy of the Tongue*' now and then occur, apparently as a sequel of malarial fever.

A writer in the '*Indian Journal of Medical Science*'¹ reported the case of an old Mussulman writer who, during recovery from a severe attack of remittent fever, six months previously, felt only slight lancinating twitches in the tongue, which gradually increased in size until it protruded beyond his lips and totally obstructed the passage of solid food. The organ presented a red shining appearance, with a soft elastic feel, as if it contained air, uniform throughout. In other respects, he enjoyed tolerable health. The part was leeches and blistered without effect. An ointment, of half a drachm of iodine to half an ounce of ung. resinæ, was rubbed in daily upon the surface which had been blistered. In ten days, the tongue was reduced to nearly its natural size.

Another contributor to the same publication² gives the case of a Brahmin, fourteen or fifteen years of age, who, about six or eight weeks previously, was attacked with swelling of the whole body, legs, and feet. This diminished for a short time, but returned and finally receded, leaving his face and tongue swelled. The tongue was greatly

¹ Vol. ii., p. 298.

² Vol. i., p. 243.

enlarged, protruding considerably beyond the lips, and separating the jaws in front for nearly an inch. The constant pressure of the tongue had caused the upper and lower incisors to protrude somewhat outwards, and to make deep indentations on its upper and under surfaces. He could not articulate, and his inability to swallow more than a few drops of fluid at a time had reduced him to a state of great emaciation. Several incisions in the protruding portion reduced the size of the tongue so much that extensive adhesion between the tongue and right cheek was detected. On the left side, it appeared as if the molars had grown into the tongue, which was firmly fixed to them and apparently to the floor of the mouth. These adhesions were separated by knife and finger. The bleeding thus caused diminished the size of the tongue, which became healthy, but the protrusion of the incisors remained until he went home; these teeth did not come into close contact when the molars met each other.

A very nearly parallel case occurred to Sir Joseph Fayrer.¹ A healthy-looking Bengal Brahmin, *ætat.* 20, said that, about ten years previously, he had a severe attack of fever and, immediately after it, his tongue began to swell. The size of the organ somewhat varied from time to time. The tongue was enlarged, not protruding beyond the lips, but the mouth could not be closed, as the jaws were separated and the lower teeth everted by the pressure. The lower jaw was pushed downwards, and the upper and lower teeth were separated by an interval of nearly an inch. The tongue was much enlarged, round, and not compressed; it had several patches of enlarged papillary growth, not unlike the commencement of epithelioma; it was increased in length about two inches, and was smooth, red, and cylindrical towards its apex. A portion of the enlarged papillary growth having been snipped off and examined under the microscope, exhibited only fibrous structure. The tongue was slightly painful on pressure, but it was the inconvenience rather than the pain that he complained of. There was difficulty in masticating, and speech was much affected. The tongue was nearly reduced to its normal state in about six weeks by bandaging and a course of iodide of potassium. The hypertrophy of the papillæ had almost disappeared, but bandaging did not affect the distortion of the lower jaw. This, it was feared, was permanent. The distance between the upper and lower incisors, when the molar teeth were in apposition, was nearly an inch.

It is noteworthy that Fayrer's patient said that he never had syphilis and never took mercury; and that there is nothing to show that the other two patients had been mercurialised.

At Bishop's College, near Howrah, a clergyman's son, *ætat.* 7, was running upstairs with a plate, fell forward, and nearly bit his tongue through. There was no bleeding when I got to him, but the anterior

¹ *Observations*, p. 537.

half of the organ appeared to be attached by little more than the mucous membrane below. Finding that, when the mouth was open, the parts lay in perfect apposition, and knowing that the child was singularly quiet and obedient, I merely bandaged the jaw and ordered fluid nutriment; but I feared exceedingly! The wound, however, healed rapidly. An elevated, semi-lunar cicatrix marked its track. Long before this occurred, Mr. W. Jackson recorded an almost exactly parallel case in vol. i., N.S. p. 630 of the 'I. M. J.'

Epithelioma of the tongue.—I know of two cases which proved fatal in brother officers of my own, and I have been consulted in the cases of two East Indian ladies, an English sailor, a poor Calcutta native, and a Bengali gentleman long resident in London.

Dr. James Cleghorn refers to ¹ three cases in which he has excised the tongue with scissors—twice after ligaturing the lingual arteries in the neck, and once, partially, without ligaturing those vessels. The patients were, presumably, native men.

One morning I found a young adult native exhibiting himself in the verandah of my hospital. He was so constructed that, when he opened his mouth widely and depressed his tongue, the epiglottis was visible from its attachment to the tongue.

Dr. Rice ² found, in a young Mussulman, a fibrous tumour between the base of the tongue and the epiglottis. It greatly obstructed breathing and deglutition. The mass, with the epiglottis, was removed by écraseur. It measured $6 \times 6\frac{1}{2}$ inches, and weighed 2 oz., 2 dr. The voice was restored and he could eat all kinds of food.

Diseases of the Pharynx.

As would be expected in a country where there are sudden and great transitions of temperature, *Sore and Relaxed Throats and Inflamed Tonsils* are of almost daily occurrence. I have repeatedly seen such extreme inflammatory swelling of both tonsils as to threaten closure of the glottis for some hours.

I saw a few cases of *Granular Pharyngitis—Clergyman's Sore Throat*, in India. There, as is doubtless the case elsewhere, clergymen and singers are very apt to imagine that their voices are becoming impaired whenever a little hoarseness, or soreness, or relaxation of the throat occurs. In such cases, doubtless, the use of strong gargles and of capsicum lozenges creates disease which would not have occurred spontaneously. I occasionally gave advice, over a long series of years, to an East Indian lady who sang in a church. The disease was slight, and was kept in check by a little care. I have always thought that this is rather a neurosis than a local organic affection, *vide* p. 389. A distinguished Indian chaplain lost his voice for a time

¹ I. M. G. for May 1884.

² I. M. G., June 1876.

so completely that he could only express himself in writing. The Rev. — was a man of singular cleverness and a very striking preacher, but he was wanting in sound judgment and was very excitable. A sea voyage completely restored his voice ; but, a few years afterwards, his health broke down under heavy duty in extremely hot weather, and he died at a distant station to which he went for change. It was said that he died from ardent fever. The station of — has long been notorious for a malarial fever of which ardency and head-symptoms are markedly characteristic. In this case, if the head affection was, in reality, a complication of fever, I apprehend that the *febris loci* attacked a faulty and almost exhausted brain.

It appears, from various passages in his letters,¹ that Dr. Duff, the great Indian missionary and preacher, was long threatened by this malady, the tendency to which was developed in his progresses through Scotland and America in 1850 and 1854, when he systematically addressed three hours' long discourses to vast multitudes in stifling halls and churches, leaving off utterly exhausted and absolutely wet through. 'I was soon in a regular bath, my very coat being wet through, the consequent exhaustion being what might have been expected.' 'It was a vapour bath, somewhat like Calcutta at the end of the wet season. I was drenched clean through, my very coat soaking through.' 'The church being much heated, I came home dripping. Throughout the night, being very restless and half awake, the enemy took advantage of my physical weakness to tempt me with wretched thoughts and horrid dreams. How I longed for the morning !' His complaint was almost always of sore throat. An American reporter wrote : 'Dr. Duff is obviously labouring under ill-health, and his voice, at no time very strong, occasionally subsides almost into a whisper.' His biographer writes, describing the close of one of his addresses : 'The thrilling voice died away in the eager whisper, which, at the end of his life, marked all his public utterances.' We have frequently such expressions as these, 'No one can ever fully know how much I often suffer, both in mind and body, in the midst of these frequent, prolonged, and violent exertions.' Then the result—'the whole frame feverish, the whole nervous system, from the brain downwards, in a state of total unrest.' The very tendency to sleep gone. Among the consequences were 'congestion' of the brain and anæmia, necessitating a long rest in Europe. In the chapter on *Diseases of the Nervous System* I have stated my opinion that the follicular changes in the mucous membrane of the pharynx in clergyman's sore throat are as little the cause of that affection as the attendant bronchitis is the cause of neurotic asthma. I believe the affection now under discussion to be a true neurosis, consequent upon overwork and over-excitement of the nervous system.

¹ *Life of Duff*, by George Smith, vol. ii. *passim*.

My friend, Dr. William Palmer, has given a very full account¹ of a European, *ætat.* 28, who was attacked at sea, on his passage from Ceylon to Calcutta, with symptoms of *Inflammation of the Fauces* and *Dysphagia*, of which the following were the chief. He was very restless and in great agony, being in constant motion, either making attempts to swallow or to steam his throat, or else walking about, moaning and groaning with the pain caused by such efforts. He complained of sore throat which prevented him from speaking, except by almost inarticulate monosyllabic sounds. The pain was referred to either side of the upper part of the larynx or hyoid bone, whence it extended to the ears. There was no marked tenderness on pressure over the painful parts. The sub-maxillary glands were slightly swollen and tender. At every attempt to swallow, he was obliged to stand or sit up; he then appeared to be preparing himself for some extraordinary exertion. A little fluid being taken into the mouth, then, with a convulsive effort, throwing the head upwards and forwards, and at the same time thrusting the arms violently in the same direction, a small quantity seemed to go down. After three such violent efforts, made at intervals of half a minute, he, being exhausted, was obliged to eject the unswallowed remainder—about half the original quantity. When water was taken, the rejected matter was almost as viscid as the white of egg. He could open his mouth, to a very small extent, with difficulty. The tongue was slightly swollen and covered with a thick, white, parchment-like skin, partly, but not altogether, caused by the use of a tannin lotion. The fauces were of a dusky red colour, and covered with a layer of transparent mucus, but not at all swollen in any part; on the contrary, the gorge appeared unusually large and open. A feather did not produce any motion or inconvenience when freely rubbed about all parts of the fauces. The finger could be pushed into the mouth and all about the fauces and pharynx without producing any reflex actions, although the patient was, in all respects, so irritable and excitable. There was total absence of the ordinary rapid ascent and descent of the larynx and hyoid bone which accompanies the natural act of deglutition. Dr. Palmer, recollecting the cases of George King and John Cavay, given in Dr. Todd's description of erysipelatous inflammation of the fauces, in his 'Clinical Lectures on Acute Diseases,' was assured that his patient was suffering from the same disease. Large poultices were kept constantly to the throat; strong solutions of nitrate of silver and, at length, the solid caustic were applied to the affected surface, and dilute nitro-hydrochloric acid, with infus. krameriæ and tannic acid, were given internally and as an application to the fauces. Under this treatment, the disease yielded in a week from the date of admission.

¹ *I. M. G.*, vol. i., p. 82.

A distinguished brother officer of my own and an English lady died of *Malignant Disease* (probably Epithelioma) of the Tonsil.

Kenneth Mackinnon cut away a large tumour, imbedded in the pendulous palate of a boy, which greatly interfered with swallowing and respiration. Hæmorrhage threatened suffocation, but was arrested by touching a large bleeding vessel by the point of a hot iron. The parts healed kindly in a few days. The mass was firm and fleshy, but he did not cut into it before he sent it to the Calcutta Museum.

Prep. D 7, in the Grant M. C. collection, is described as 'tumour, apparently malignant, attached to the palatine arches near the tonsils; a prolongation of it entered the larynx.'

My father told me that, when cruising on the coast of some leech-producing country, the ship's butcher brought him several 'lively' leeches which he had taken from the fauces of oxen purchased on shore. Cases in which leeches were lodged in the nasal passages will be found in the chapter on *Diseases of the Nose*, p. 474. In July 1840, Mr. Healy reported from Candahar, to the Medical and Physical Society of Calcutta, four cases of hæmorrhage from the mouth, caused by *Leeches found in the pharynx, posterior nares, palate and ears*, where they appear to have attached themselves after the persons had drunk some muddy water. In three cases, the leeches were readily discovered on examining the mouth. They were found adhering to the palate and the pharynx. The presence of a leech in the fourth was suspected from a slight oozing of blood down the pharynx, for it was out of sight. Salt and water was injected through the nose, and the leech was ejected from the mouth some hours afterwards. The leeches were of moderate size, resembling the medicinal leech.

I have published, in my 'Medical Jurisprudence for India,' p. 619, several cases in which *Living Fish were impacted in the Glottis or Pharynx*, causing or threatening suffocation. For cases in which fish entered the air passage, *vide antea*, p. 508.

Sir Joseph Fayrer has most kindly sent me his experience of one of these cases. A child of 3 or 4 was brought to the Calcutta M. C. Hospital dying of suffocation. It had been playing with some little fish (*anabas scandens*) in a basket; they were alive and kicking, and, somehow, one of them went head foremost into the child's mouth and throat, and could not be got back on account of the spines of the dorsal and other fins. It was got out with some difficulty, but the child could not be resuscitated. The fish was in the gullet, but the pressure closed the glottis.

The following cases did not appear in my work on Medical Jurisprudence. Dr. Dantra¹ was called to a Madras sepoy. While he was swimming with his mouth open, a fish, of some 3 × 1 inches, entered his mouth and seized the uvula. Pulling was abandoned as the gills

¹ *I. M. G.*, September 1878.

and scales caught upon the dorsum of the tongue. The head being pressed laterally with dressing forceps, it let go.

It was reported, in 1829,¹ that a lusty Chinese at Malacca, being angry at having caught only one *ikan lidah*—tongue-fish or sole—bit the fish, which made a plunge forward and stuck in his throat. He was conveyed to the fish market, where the fish was extracted by means of a pair of pincers, with considerable effusion of blood.

Dr. T. A. Wise reported one of these cases as it occurred to Mr. Taylor.² A native infant, ten months old, whilst crawling on the floor, put a fish into his mouth, and his mother was unable to extract it. This occurred a quarter of a mile from the hospital, and the child was apparently dead, being quite insensible, its face livid, and its respiration and circulation suspended. A bulky substance was seen closely impacted in the back part of the fauces and immediately over the glottis. Ineffectual attempts at removal having been made, laryngotomy was performed, and respiration was restored. The fish appears to have been merely lying over the fauces, as it was easily extracted by means of long forceps. It proved to be a species of sole called *Pan* (*Pleuronectes Pan*), and was five inches long. Recovery was complete.

Sub-Assistant-Surgeon C. K. Mitter had³ the case of a boy of five, who placed in his mouth the head of a living fish, *Kholesa Mách*, which made its way into his pharynx. This fish had sharp dorsal spines directed upwards and backwards. It was shaken away with considerable difficulty. It was $3\frac{1}{2}$ inches long and $1\frac{1}{4}$ inch broad.

The 'Homeward Mail,' of June 30, 1873, gives a report of an inquest held at the General Hospital, Madras, on a native man, who, when catching fish with his hands in a shallow tank, placed the head of one, about 6 inches long and $1\frac{1}{2}$ inch thick, in his mouth and bit it, when one of the sharp points in the back fin stuck in his hand. He cried out, and the fish jumped into his throat and stuck there. Dr. Liston Paul 'found that the only means of getting at it was by making an opening in the neck. The deceased was much exhausted, and died as soon as the operation was performed.'

Mr. R. G. Mathew, of Midnapore,⁴ was called up in the night to see a man who was lying in the verandah, having been brought in twenty miles from the country. He was quite comatose, and covered with cold perspiration; the teeth were firmly clenched, the pulse was almost imperceptible, and the breathing was most difficult, and accompanied with a peculiar stertorous sound. He had been fishing and, a fish having got out of his basket, he put it between his teeth, intending to kill it; but it escaped into his throat. The tail could be seen pointing upwards, and could be barely reached with the forefinger.

¹ *Asiatic Journal*, vol. xxvii., p. 509.

² *C. M. T.*, vol. ix., part i., p. 154.

³ *I. M. G.*, April 1875, p. 99.

⁴ *I. M. G.*, November 1872.

It had been dead for some hours, and emitted a most offensive odour. The accident had occurred about twelve hours previously. It was, with considerable difficulty, removed entire by forceps guarded with lint; the dorsal fin, armed with several sharp points, had severely lacerated the back of the pharynx and the soft palate, and had entirely torn away the uvula. It was a Koiee Mach (*Anabas scandens*), or climbing perch. The patient remained in a precarious condition for some little time, but returned home convalescent in a few days. The Assistant-Surgeon told Mr. Mathew that a child, related to himself, was nearly choked in the same manner; but the fish was promptly removed by blacksmith's pincers.

Prep. 87, Series 18, in the Madras Collection, is 'A sole fish removed from the gullet of an infant about seven months old.'

In this book and in the 'Medical Jurisprudence for India,' I have now cited sixteen examples of this curious but evidently by no means very rare accident. I am informed by Dr. Francis Day that it is usually the *Anabas scandens* who thus embarrasses himself and his fisher friends by the climbing 'ambition which overleaps his selle and falls on the other side.' It appears that, in this accident, instantaneous death must be caused (1) where the head gets through the rima; (2) or completely covers the glottis; and (3) where it compresses the back of the larynx. It is clear that, where death is immediately threatened, and the lodgment of the fins and gill-plates prevents rapid extraction, the trachea, not the larynx—which the fish, in its active climbing, may enter or cover at any moment—ought to be opened.

My friend Dr. Day, who is practically well acquainted with this class of accidents, tells me that we may generally relieve these cases of pharyngeal obstruction—where the fish is not so large as to kill by compressing the back of the larynx—by taking long scissors and forceps, and cutting out as much of the fish as can be reached. It is at once killed. If the head will then pass down the œsophagus, well. If not, it will quickly putrefy and pass.

An eminent clergyman in Calcutta was in great danger from swallowing artificial teeth which lodged in the œsophagus.

I saw or heard of a very few cases of Quinsy in Europeans and natives.¹

In his highly valuable work, 'An Epitome of Reports of Medical Officers of the Chinese Customs service' (page 179), Dr. Gordon cites the following: 'At Newchang, during the winter of 1880-81, quinsy attacked nearly every foreign adult in the settlement. The

¹ Stories, of the usual type, of the cure of quinsy by laughter are current in India—of an officer, left by his servants to die, in whom the antics of a monkey excited salutary muscular action; and of another who heard his companion desire the table servant to bring a mare (*murdwan*) instead of a kid (*hulwaun*) for the day's repast.

majority of the patients had more than one attack; in some instances, the disease was very severe. The largest number of cases and the most severe occurred in the first half of the winter. After a considerable fall of snow, the cases decreased in number and severity. 'It is a common matter of observation that simple tonsillitis occurs epidemically, particularly in cold and damp weather, but *Epidemic Quinsy* is, I think, rare. I find, in an account of the 'Distempers incident to the Virgin Islands of America,' in the 'Gentleman's Magazine' for February 1752, an expression which appears to imply that quinsy occurred there epidemically. 'Quinsies, peripneumonies, and pleurisies do most commonly reign in April, May, and June.'

Dr. Dubansquet Laborderie gives ¹ cases of quinsy occurring in unhealthy houses, in which infection was distinctly traceable. The tonsils, velum, and pillars of the palate were swollen and red (this is scarcely quinsy, which is *suppurative tonsillitis*) but free from diphtheritic exudation. Deglutition was difficult. The editor of the Journal suggests that these cases resembled an imperfect form of scarlatina.

A young English married lady in Calcutta, of rather strumous appearance, died of *Post-Pharyngeal Abscess situated low down*.

Diseases of the Œsophagus.

In a remarkable case, described under the head of aneurism, at page 495, a man lived for more than a year after the occurrence of great hæmorrhage and discharge of laminated clots from the sac of an aneurism of the aorta which opened into the œsophagus. He died from another disease.

The three cases which follow illustrate *the tendency which malignant disease of the pharynx and upper part of the œsophagus has to involve or to open into the air passages*.

Preparation 375 in Ewart's Catalogue displays *Malignant Disease of Pharynx and Œsophagus, involving the Larynx*. There is malignant ulceration of the left vocal cords, and several nodules of medullary matter are deposited beneath the mucous membrane of the lower part of the larynx. The patient, a Portuguese half-caste, could not swallow for some days prior to his death. Neither could a tube be passed, owing to œsophageal obstruction. Respiration was so seriously embarrassed that tracheotomy was performed eleven days before he sank.

Prep. 5, Series 8 in Sibthorpe's Catalogue is œsophagus exhibiting stricture about $2\frac{1}{2}$ inches above its lower end. The extent of tube involved in the disease is nearly $3\frac{1}{2}$ inches. The left bronchus is adherent to this portion of the œsophagus and the canals communicate by a sinus.

¹ *Bulletin Général de Ther.*, January 1886, and *Provincial Med. Jour.* Feb April,

Mr. A. K. Lindesay had a patient, a sergeant, *æt.* 55, in whom there existed a *malignant stricture of the œsophagus*, a good way above the cardiac orifice, occupying four inches of its canal, the whole circumference of which was found thickened and ulcerated. There was an opening in the back of the trachea communicating with the diseased part of the œsophagus. It was irregularly circular, about as large as a shilling, and was situated about half way between the cricoid cartilage and the bifurcation. He survived the occurrence of this communication, with occasional attacks of violent coughing, for about 19 days.

I met with two cases (native) of *Stricture, doubtless malignant, of the Cardiac Orifice of the Stomach*. Each of these unfortunates could gulp down precisely half a pint of milk, the quantity which the œsophagus was able to contain. In the course of a few minutes, nearly the whole of the fluid was eructated forth by repeated expulsive contractions of the œsophagus, causing great distress.

Prep. 150, 8th Series, Madras Collection, is from a European pensioner suffering from dyspeptic symptoms of a year's duration—sense of epigastric pain, discomfort in swallowing solids, pain in left shoulder, and in the course of the œsophagus, irregular bowels, flatulence and salivation. He died after 10 months' stay in hospital. There is a large fungating cancerous growth involving the lower end of the œsophagus and cardiac portion of the stomach. The bronchial and lumbar glands were infiltrated with the same growth.

Transposition of the Abdominal Viscera.

Mr. Raleigh¹ reported the post-mortem examination of a man in whom there was complete *transposition of the contents of the abdomen*. Nothing is said with regard to the position of the thoracic viscera.

In Prep. C 11, in the Grant Medical College Museum, Bombay, all the viscera of the thorax and abdomen are transposed except the large intestine, the cæcum and sigmoid flexure retaining the usual position.

Dr. A. Garden examined the body of an outwardly well-formed, plump, and healthy-looking still-born native female infant, in which the spleen, stomach, small intestines, a portion of the colon, and the left lobe of the liver occupied the left side of the chest, the left half of the diaphragm being deficient. The left lung was 'small, undeveloped, brownish-red in colour, seemingly normal in structure, about the size of half a walnut.' The heart was much to the right, the aorta arching from right to left. The child had never breathed.² In his excellent remarks on 'the value of systematic examination of still-born children,' read at the Medico-Chirurgical Society in March 1884,³ Mr. J. H. Sutton describes 'a foetus with congenital deficiency of one half of the

¹ *I. M. J.*, vol. i. p. 36.

² *I. M. G.*, vol. v. p. 55.

³ *M. T.*, vol. i. for 1884.

diaphragm. The left lobe of the liver passed into the pleural cavity, the lung of that side being in a very rudimentary condition.' Such is the normality of many developmental 'abnormalities.'

A great number of very remarkable cases of *Abdominal Wounds* occur in our local medical literature. Young lads, employed in herding buffaloes, are frequently gored by the half-wild animals. A boy, who had been injured in this manner 3 or 4 days previously, was brought to my dispensary at Chittagong. A lake-coloured globular mass, which appeared to consist of the whole of the small intestines, lay upon and entirely covered the abdomen. A coat of inflammatory exudation completely glued together and coated the coils of intestine, the outlines of which bulged under it. The gut had escaped through a small wound in the abdominal wall beside the navel. Every other moderate attempt to reduce the inflamed and greatly congested mass having failed, I, perceiving that atmospheric pressure upon the abdominal wall prevented the return of the bowel, placed him, lying on his back, upon a bench, and curved my little finger (no other finger could enter the abdominal cavity) into a hook: with this, standing up, and exerting all my strength, I, meeting with great resistance, raised the parietes of the abdomen. The mass was then reduced by gentle pressure. Although the bowel had been so long exposed, and had been so generally inflamed, no peritonitis or symptom of other disorder followed in his rapid recovery. There was a little healthy suppuration as the wound was healing.

Dr. Spilsbury recorded¹ the case of a stout healthy grass-cutter gored by a spotted deer. The horn appeared to have passed just over the spermatic cord on the left side, entering the inguinal canal and penetrating the cavity of the abdomen nearly in the direction of the canal. Some ten or twelve inches of the small intestine protruded. The wound was so very small that it was necessary to enlarge it before the hernia could be returned. Some tenderness of the abdomen, difficulty in passing urine, and fever ensued, and were readily put down. On the third day, the wound was united, except at the stitches.

I was called to see a poor native boatman, at Chittagong, whom I found upon the point of death. As he was cleaning his boat, a crocodile (*muggur*) came stealthily along under water and seized him. His companions, holding him, tugged against the animal, which only let go when it had torn away nearly the whole of the abdominal parietes.

Mr. Samuel Brooking² relates the case of a Rajpoot Mahratta, a free liver, *æt.* 33, who, having attempted to murder his wife, ripped up his own abdomen with a blunt clasp knife. The jagged wound was three inches long at the peritoneum and much larger externally. Through it, the enormously distended stomach, its outer coat lacerated

¹ *C. M. T.*, vol. vii., part ii., p. 467.

² *Madras Quarterly Journ. of Med.*, vol. ii. p. 356.

in many parts, and a portion of adhering omentum, protruded, being more than a foot in diameter. The reduction was difficult. Some abdominal pain and tenderness ensued. He was discharged well in a month and nine days.

Mr. Hinder reported¹ the very similar case of a Calcutta sweeper, about 40 years of age, said to be vicious and intemperate, who, having murdered his wife, endeavoured to commit suicide. He was found to have two incised wounds completely dividing the anterior abdominal wall, one extending obliquely downwards from the ensiform cartilage, about $2\frac{1}{2}$ inches, the other, transverse, about $1\frac{3}{4}$ inch long, above and to the left of the navel. Through this latter, about 2 inches of omentum protruded. He was much collapsed on admission. There was some peritonitis which was easily subdued. On the morning of the fifth day, he coughed and strained so violently that the lower wound opened and about 6 inches of small intestine protruded. Dr. Baillie reduced this. He recovered with difficulty, but was sent to jail convalescent in a month and ten days.

A native lad, about 18 years old, received a spear wound in the epigastrium. Sixteen days afterwards, Mr. Odevaine found a long fleshy substance, of a pale pinkish colour, lying outside the parietal wound. It measured 5 inches in length, and was from $1\frac{1}{2}$ to 2 inches in diameter. It was rounded, was not lobulated, but had a rough granulated appearance. Finding that it could not be returned, Mr. Odevaine ligatured it. The whole had separated on the tenth day after the operation. Four days later, the patient absconded, having merely a deep ulcer, of the size of a shilling, on the site of the wound. No communication with the abdominal cavity could be detected. Mr. Odevaine considered that this was a protrusion of the tail and body of the pancreas.² Dr. Kenneth Macleod³ combated this opinion, denying the probability of the idea that the pancreas was, in the above case, or could be, protruded through a wound in the anterior abdominal wall. He reported a series of dissections and manipulations which he had made in support of his view, and argued that the protrusion was omental. Dr. W. B. Beatson reported⁴ a case which had occurred in his own practice, in which a mass of omentum, protruded from a gunshot wound 'on the lateral aspect of the left side of the thorax,' strongly resembled the pancreas. Mr. Adam Taylor⁵ had a case of suicidal abdominal wound in which he considered that the tail of the pancreas protruded; but it does not appear that, as the editor of the 'Indian Medical Gazette' remarked, the histological characters of pancreatic structure were identified by Messrs. Odevaine and Taylor.⁶

¹ *I. M. G.*, vol. i. p. 9.

² *I. M. G.*, vol. i. p. 183.

³ *Ibid.* p. 301.

⁴ *Ibid.* p. 329.

⁵ *Ibid.* 258 B.

⁶ This volume of the *Gazette* contains some other valuable cases of abdominal wound.

In a patient of Baboo Sreenauth Mookerjea,¹ a portion of lung protruded through a wound in the right hypochondrium. It was 4 inches long and 2 broad. It was tied close to the abdominal wound. As the mass contracted daily and loosened the silk, fresh ligatures had to be applied repeatedly. It took seven weeks to separate. No bad symptom followed the application of the ligature.

Diseases of the Stomach.

Gastritis, as evidenced by a fiery red sharpened tongue, and by great irritability of stomach, is frequent in both Europeans and natives dying of abdominal dropsy, especially when there is kirrrosis of the liver. What is designated as a case of idiopathic acute gastritis, in a *temperate* European, *æt.* 25, is given by Mr. E. W. Eyre.² The patient had been complaining of abdominal pain for some days previous to admission. He had been taking Morrison's pills and copaiba and iron for gonorrhœa, and an emetic a few days before. There were severe abdominal pains, chiefly in the epigastrium and across both hypochondriac regions, and much tension of the abdominal muscles; no swelling or increased temperature, pulse rapid and small (he was much alarmed), skin cool; tongue clean. In the evening, the pain, which had been relieved, returned. Lay on the side groaning; stomach irritable, pulse rapid and small, cold sweats, thirst and rejection of fluids swallowed. He passed a bad night, restless and moaning, face flushed and swollen, stared wildly about, not replying to questions, stomach irritable, pulse rapid and sharp. A tense painful swelling extended across from one hypochondrium to the other. He brought up some dark-coloured fluid, became exhausted and sank. *P.M.*, Stomach greatly distended. Its mucous membrane and that of the duodenum was highly vascular. The stomach and duodenum were filled with thick dark-coloured fluid similar to that which had been vomited. Some of this, mixed with mucus, adhered to the mucous membrane. There were firm (old) adhesions between the stomach and spleen and left lobe of the liver.

An intemperate sergeant³ who had, for several days, been drinking ardent spirits to excess, was admitted complaining of constant vomiting, great thirst, and acute pain, and a sense of burning in the epigastrium. There was extreme debility, a remarkable anxiety of countenance, restlessness and a continued tossing of the arms. Pulse not perceptible, skin cold and clammy, bowels costive. Towards evening, the stomach was not so irritable, but still rejected all fluids. In the night, he died insensible. *P.M.*, Stomach very red and highly

¹ *I. M. G.*, vol. v. p. 56.

² *I. A.*, No. 3, p. 452.

³ *Madras Quarterly Medical Journal*, vol. iv. p. 352.

congested; the liver was rather increased in size and soft. The kidneys were enlarged, but not otherwise diseased.

Prep. 380 in Ewart's Catalogue represents 'Chronic gastritis occurring in a European, a habitual drunkard.' There is thickening of the mucous membrane, which is arranged in longitudinal folds, many of which are a line and a half in height. These appearances are most marked along the line of the greatest curvature. The peritoneal tunic is thickened and opaque.

Preps. 379 and 381 are also excellent illustrations of the effects of chronic gastritis.¹

Gastrodynia.

My friend Dr. Thomas W. Wilson has published² an analysis of cases designated by the natives of Bengal *Peet Sool*, or *Peet Bedna*. The main characteristics of this condition are indigestion, pyrosis, and gastrodynia. Dr. Wise, who wrote on this subject in the second number of the 'I. M. J.,' attributes the disease to a nervous state, analogous to neuralgia or asthma. Dr. Wilson, upon full inquiry, considered that *Peet Sool* 'arises from no peculiar diseased condition.' Some of the cases which he examined proved to be colic, passage of gall-stones, or indigestion. He gives an account of the post-mortem examinations in four cases. In these were found (1) an ulcer the size of a four-anna piece (sixpence), which had given way, causing fatal effusion. 'The ulcerated opening appeared as if a bit had been punched out; ' (2) a perforating ulcer in the duodenum, not apparently malignant. Effusion into the peritoneal cavity; (3 and 4) in these cases there was malignant disease of the pylorus. In Dr. Wilson's third case, the poor man, worn out by suffering, hanged himself. In my 'Medical Jurisprudence for India' will be found (p. 667) details of other cases in which natives, suffering from *Peet Sool*, committed suicide. Dr. Wilson mentions that this affection is attributed to a weapon in the hands of Siva; and, though the deity inflicts the blow, he cannot remove the disease. Consequently, the sufferer often despairs, and seeks relief by suicide. I was told by Mr. W. J. Ellis, then of Pubna, that he considered this affection depended on excessive fish-eating, especially of Hilsa fish. It prevails among all classes of natives, from the richest to the poorest. It had almost disappeared from the Pubna Jail since the use of Hilsa had been discontinued. He thought that, at any time, he could find a hundred cases in Pubna. He had never discovered any satisfactory explanation by post-mortem examination. He had seen several cases in which the patients had destroyed themselves. In 1823, Mr. Andrew Stirling mentioned that,

¹ See Dr. Kyle Cotter's 'Observations on Acute and Chronic Gastritis of Hot Climates,' *I. M. G.* for October 1884, p. 279.

² *I. A.*, No. 3, p. 97.

during four recent years in which he witnessed the great ceremony at Jaggernaut, only three cases of self-immolation occurred. One of these might have been accidental; the other two victims had long been sufferers from excruciating complaints, and chose that means of ridding themselves. I had a patient, in the jail of Purulia, who used to sit, evidently in great agony, from which he sought relief by pressing the pit of his stomach against a log of wood. At his death, I found very great contraction of the pylorus, by sub-mucous deposit. The mucous surface, at the strictured part, appeared perfectly sound. At p. 802 of my 'Medical Jurisprudence' will be found cases in which persons, alleged to be the subjects of this malady, committed homicide; especially one in which a man killed a little girl with a piece of wood for sitting on, called a *pinrah*, which he was in the habit of pressing on his stomach when the pain was excessive.

Doubtless, both Dr. Wise and Dr. Wilson are right in their views upon this class of disorders. The rice and fish-eating denizens of Bengal are liable to various kinds of belly-ache, among the principal causes of which are colic and organic diseases of the stomach.

Simple Perforating Ulcer of the Stomach.—This disease must be rare in India. I saw it but once, and I know of only three other recorded cases. The specimen which I sent to the College Museum is thus described by the late Dr. Collis: ¹ 'In this specimen, taken from a patient who died of peritonitis, is a perforating ulcer in the upper and posterior part of the stomach, which is adherent to the inferior surface of the right lobe of the liver. It is about the size of a shilling; the opening into the peritoneal cavity is somewhat oval in shape, and partly protected by a valvular arrangement. The edges of the ulcer were a good deal thickened, and the adjacent surfaces of the liver and stomach were covered with recent lymph.'

Mr. B. Wilson ² attended a European gentleman, at Bancoorah—the length of whose residence in India is not stated—who died from peritonitis resulting from the sudden separation of an ulcer of the pylorus, at its junction with the duodenum, from the under surface of the liver, to which it had been adherent. The coats of the pylorus at its junction with the duodenum, to the extent of about two inches, were very much thickened, and the passage so contracted as to be rendered nearly impervious. The ulcer was oval, with regular round thickened edges, and easily admitted the index finger. He had complained, for several days, of a peculiar sinking sensation in the stomach, 'as if his stomach was quite gone,' and sometimes he felt as though a heavy weight was pressing on it.

There is a case by Dr. J. Carmichael ³ in which an ulcer 'circular

¹ *I. A.*, No. 21, p. 294.

² *I. M. J.*, vol. ii. p. 47.

³ *I. M. G.* for October 1871, p. 212.

and regular, as if punched out,' was found in the stomach of a sepoy, *æt.* 39, who never had hæmatemesis. See a case by Dr. Wilson, at p. 549.

The cases of *Malignant Disease of the Pylorus* which came under my care in India did not differ, in any important particular, from those which occur at home. I think that more than is done at present might be effected, in these cases, by the use of nutrient enemata—*without salt*, which irritates the bowel.

In speaking of the diagnosis of hepatic abscess, I have mentioned the fact that it is not always easy to differentiate cancer of the pylorus from some cases of that disease.

Assistant-Surgeon D. D. Sen relates¹ the case of a Mohammedan opium-eater, *æt.* 50, in whom there was a hard cancerous ulcer, about the size of a closed fist, in epigastrio, which had existed for two months. From this, undigested food escaped. He left in three weeks, apparently wishing to die at home.

Preps. 384 to 389 in the M. C. Collection illustrate malignant disease of the stomach. The last is from an Australian horse. A foreign growth (melanotic?), about the size of a walnut, is situated between the mucous and muscular tissues.

I have already spoken (pp. 226 and 250) of *Hæmatemesis* as occurring in splenic disease. In 1855, I published² reports of two cases of fatal hæmorrhage from the stomach. The subject of the first was an East Indian, *æt.* 47, who apparently lived freely and held a position which demanded great exertion and exposure in the open air. Some years previously, he had suffered much from intermittent fever with splenic enlargement; but, when I saw him, there was no outward evidence of splenic or hepatic disease. The conjunctivæ were markedly yellow. Nine months previously, he was again attacked with fever and had remained weak, languid, and easily exhausted ever since. From that time, he became liable to frequent and profuse epistaxis, which, however, had not recurred of late. He did not complain of any uneasiness about the gums, but his breath was very offensive, and his wife noticed that saliva tinged with blood discoloured his pillow every night. At ten, one night, he vomited a large quantity of clotted blood, and had a restless night. In the morning at daylight, he again vomited blood in considerable quantities. After this, there was no hæmorrhage. I prescribed astringents and antacids and a large enema. I could not prevail upon him to lie by, and I saw him about in some of the hottest days of an unusually trying season. At the end of a month, having dined very heartily, after which he had felt dull and oppressed, but, before going to bed, had sat in the verandah exposed to a rather chill air, he vomited, at midnight, a great deal of blackish matter and afterwards large quantities of blood. The vomiting of dark blood and of bloody fluid containing clots continued unchecked by astringents and ice.

¹ *I. M. G.* for January 1884, p. 19.

² *I. A.*, No. 3, p. 457.

Latterly, dark, pure clotted blood, which coagulated in the vessel, was alone ejected. An immense gush occurred at about six in the evening, and he expired at once. A *p.m.* was not allowed.

A European of spare make, about thirty years of age, was seen by me occasionally for three years and a half previous to his death. He was avowedly intemperate, and his nervous system had become greatly shaken. I attended him for one of those gastric and hepatic attacks which generally result from excess in that climate. He then had a peculiar form of petechial extravasation, in large irregular patches of a dirty claret colour, and nearly as large as the palm of the hand, over various parts of his otherwise sallow skin. Subsequently to this, I treated him in a very severe attack of cholera. For a month or six weeks previous to his death, he had been in the habit of passing bloody motions, probably resulting not from dysentery, as was alleged, but from passive hæmorrhage. Returning on foot, after a late dinner with a convivial friend residing at a distance of about a mile from his own house, he was attacked with vomiting of dark blood. The sub-assistant surgeon was immediately summoned and found him *in articulo*, vomiting dark blood in immense quantities. There was no autopsy.

I assume that the deaths of these two individuals probably resulted from similar trains of diseased actions. I have only known sudden and rapid death occur in hæmatemesis in cases where ulcerative action had extended from the tissues of the stomach to either the gastric or the splenic artery. The reason for believing that such a lesion had not occurred in either of the above cases is self-evident. Nor can it be considered that, in either individual, an aortic aneurysm had opened into the œsophagus. Portal obstruction—whether remote or immediate, whether dependent upon cardiac impediment, upon kirrrosis or other lesion tending to embarrass the capillary circulation of the liver, or even upon impediment within, or complete occlusion of the portal vein itself—very seldom indeed, if ever, tends to destroy life by hæmorrhage from the stomach. Rupture of the smaller vessels of the stomach, when it occurs, as it so frequently does under these circumstances, merely appears to exert, for a time, a salutary influence in freeing the obstructed vascular tract from otherwise insupportable distension. Great obstruction or complete obliteration of the main trunk of the portal vein is well known to be liable to induce hæmatemesis or melæna; still, I know of no case in which suddenly or rapidly fatal hæmorrhage from the stomach resulted from portal obstruction. Considerable hæmorrhage from the stomach or bowels may occur at any stage of such obstruction; but it appears most liable to be occasioned early, before the imperfect anastomosis has been established, or dropsical effusion has taken place. In neither of the cases related above, were the characteristic signs of narrowing or obliteration of the portal vein observed. The tendency to hæmorrhagic action, from

cholæmia or uræmia, in persons of intemperate habits is, in itself, scarcely sufficient to account for sudden death from gastric hæmorrhage in any case. In a rather large experience, I have learnt nothing to show that mere intemperance can lead to death from simple hæmatemesis. In drunkards, coffee-ground vomiting is not always a fatal symptom.

It will be observed that there existed a striking similarity in the constitutional states of these two individuals as well as in the physical causes which appear to have determined the fatal attack of hæmatemesis in each. They were both very free livers, exposed to considerable vicissitudes, but accustomed to most of the comforts and to some of the luxuries of life; they resided in malarious situations, and had both suffered from premonitory attacks of passive hæmorrhage. In each, the fatal attack appeared to be the direct result of exposure to a chilling air, when the stomach was loaded with food and probably with strong drink. I do not think that either patient was scorbutic. Here cholæmia and uræmia were probably co-existent. There was paludal cachexia, with hepatic kirrrosis or other obstructive lesion. It is evident that a chill to the surface, operating when the vascular action of the gastric mucous membrane had become excited in commencing digestion, determined the fatal outburst.

In England, I saw two cases in which perforating ulcer of the stomach destroyed life by *Arterial Hæmorrhage from* (1) *the Gastric, and, (2) the Splenic Artery.*

A sepoy who was admitted to Dr. Fairweather's hospital complaining of pain, and tenderness in the region of the stomach, said that, when lying quietly in bed on the previous night, he vomited a quantity of blood. As there was no evidence of disease about him, this was doubted. Some ten days later, however, the hæmorrhage recurred, and blood appeared in large quantity in his stools. This continued for three days, when a piece of a leaden bullet, about the size of a large kidney bean and weighing 122 grains, fell into the utensil. It was then learnt that, about 15 months previously, at the relief of Lucknow, he had been struck by a ball on the chest about an inch and a half above the right nipple, where the scar was still plainly visible. He appeared to recover rapidly and without symptoms of any internal injury. For ten days after the passing of the lead, he appeared to be recovering, and was about to go home to recruit his strength when hæmatemesis set in, worse than before. He continued for four days to vomit and to pass huge quantities of blood by stool, and then died.¹ The preparation from this case is numbered 383 in Ewart's Catalogue. The bullet appears to have become lodged in the substance of the pancreas, where its pressure caused the formation of an *Aneurysm* about as large as a pigeon's egg, communicating with one of the

¹ *I. A.*, No. xiii. p. 322.

branches of the *Pancreatic Artery*. Hæmorrhage from the aneurysmal sac had occurred, through a small ulcerated opening, into the cavity of the stomach.

In the dissecting room at Guy's I saw enormous *Dilatation of the Stomach of a rice-eating Chinaman*. The stomachs of Bengalee rice eaters are large. Fond mothers pat the prominent bellies of their children when they have eaten a full meal, and chuckle at every eructation, which is rejoiced in as the sign of a good digestion; but the stomachs of Bengalees are not usually much dilated, probably because the *marsalla*, eaten with rice, contains an abundance of capsicum. When on the long sea voyage to India, in medical charge of troops, I was called in the night to a non-commissioned officer who was roaring from pain in a much over-distended stomach. He had made a large meal upon plain boiled rice, a food to which he was not accustomed. In his paper upon Sudden Death, Brigade-Surgeon William Curran mentions the case of another soldier 'who had partaken largely of rice and rum at supper. Fermentation took place, and he died miserably during the night of rupture of the stomach.' I once, when much heated, rapidly drank off the contents of a bottle of ginger-beer with all its gas. For a moment, I anticipated sudden death. The shock produced by the backward pressure of the distended stomach upon the ganglionic plexuses was so great that I almost realised the popular legend of the young lady who died from drinking iced water when overheated by dancing.

An account is given by Dr. J. Clarke¹ of two cases of what he regards as *Spontaneous Rupture of the Stomach*. The deaths were both rapid. Dr. Clarke is fully convinced that the lesions discovered were not due to post-mortem digestion of the walls of the stomach by the gastric juice; but he does not appear to have established his position.

Sarcinæ Ventriculi were recognised in India soon after they were first seen by Goodsir in 1842. An account of a very interesting series of cases, in natives, by the late Dr. A. Simpson, then of Mozufferpore, will be found in the 'I. A.,' No. 2, page 599.

At a meeting of the Epidemiological Society in February 1884, my friend Brigade-Surgeon Scriven said that he had been sent for to a Lahore native, who was dying, it was alleged, of cholera. The autopsy showed that the alimentary canal was stuffed, almost throughout, with the seeds of *Ispaghul* (*Plantago Ispaghula*). These seeds, bruised, yield a demulcent and mildly astringent mucilage much valued by the natives in diarrhœa. Having been attacked with this malady, the poor man allowed his friends to 'throw in' the remedy so freely as to kill him.

¹ *I. M. G.* for August 1885.

Foreign Bodies in the Stomach.

There is a case,¹ by Mr. A. Gilmore, of a native who appears to have swallowed fifteen rupees about six years before his death. He complained of 'the anomalous affection vernacularly called *Durd-i-sool*, which is characterised by agonising pain at the pit of the stomach, recurring in paroxysms, only to be alleviated by strong external pressure' (see page 550). He used always to have beside him a pointed stick, on which he leant or pretended to lean very forcibly during each attack. He gradually sank with diarrhoea, having latterly seldom complained of the gastric pain. The stomach was much contracted and thickened. Nine rupees were placed, *en rouleau*, near the pylorus, close to which there was a circular ulceration with deep indurated edges. The peritoneum gave way here, when the stomach was moved, and the liquid contents escaped. He was believed to have passed the other six rupees by stool.

Mr. G. A. Watson² relates the case of a Sowar, *æt.* 23, who, using a piece of brittle willow as a *dantum* or tooth stick, and thrusting it back into his pharynx to clear away phlegm, broke it and swallowed a portion. For about three weeks, he appears not to have suffered much inconvenience, but he then got fever and tenderness in the hepatic region, followed by jaundice. He passed a large quantity of blood from the bowels and died forty-six days after he had swallowed the stick. The foreign body, which was six inches long and a quarter of an inch in diameter, was found lodged in the duodenum. One end of it projected through the coats of the intestine into the under surface of the liver, which was inflamed and suppurating.

Dr. Hutchinson³ had a similar case:—an old woman, after cleaning her teeth, passed the *miswāk*, as the natives generally do, far down to clear the fauces of mucus by retching. The stick became impacted and passed down. Three days later, she came suffering from most painful and urgent dyspnoea with frequent cough. The trachea was opened, but she died unrelieved and asphyxiated. The *miswāk*, nine inches long, was found lying in the oesophagus and 'resting on the lower margin of the stomach,' where was a small patch of congestion. The lungs were stuffed with mucus, presenting the appearances to be expected in death from acute bronchitis.

Dyspepsia.

If India could have the advantage of the presence of Dr. Clifford Allbutt, that admirable observer would find ample opportunity of

¹ *C. M. T.*, vol. vii., part ii., p. 470.

² *I. M. G.*, vol. iv. p. 117.

³ *Ibid.* p. 141.

differentiating that often ill-discriminated mass of organic lesions and functional neuroses known there under the coarse denomination of '*Indigestion*.' I think that it may be stated generally that simple dyspepsia, uncomplicated with grave organic disease, is much rarer in Europeans in India than it is at home. There are many Indian cases in which the stomach performs its work with difficulty; this especially occurs in Cholera, Chronic Diarrhoea, and Sub-Acute ('Chronic') Dysentery, and in Hepatic Abscess and other grave organic diseases of the liver. We still await an explanation of that mal-assimilation which leads to calculous disease in the sour bread-eating London child and the *chupattee*-fed peasant of the Upper Provinces, but which assumes another and more destructive type, *Morbus Bengalensis*, in the rice-consumer of the Delta of the Ganges.

Indigestion is one of the very gravest complications of Phthisis in India. He who, either in India or at home, acquires, by spirit drinking, chronic gastritis, duodenitis, and kirrrosis of the liver, of course seriously impairs his appetite for food and his digestive powers. A hardworking, ill-fed native, the victim of splenic cachexia, can hardly enjoy sound digestive power. Forty-six years ago, that valuable man Moodoosoodun Goopto, who first induced Hindoo students to dissect the human body, said what may be said with equal truth to-day: 'Nearly two-thirds of the native population of Calcutta have dyspepsia. It produces debility which predisposes to other diseases, diarrhoea, dysentery, and piles.' I have spoken of several of these stomach difficulties elsewhere. We used, formerly, to hear a great deal of loss of appetite caused by the exhausting effects of Indian heat. This may still be noticed in cases of cachexia and lowered innervation; but, when men in India are temperate over night and earn their *hazirees* before they eat them, there will be very little complaint of want of appetite in the morning. I may here make a remark which will be useful to strangers in India. One morning, at Chittagong, when I was much out of health, my native doctor brought to me, on my visit to the jail, a prisoner who had smashed a middle finger in a *dhankee* for husking rice. It occurred to me only that the case was a very common one, when, suddenly, I found that I was about to fall over the patient in a faint. I just managed to get into the open air, which revived me. I then recollected that I *had come out without food, which no one should ever dare to do in India*. After a meal, I returned and amputated the finger. This was the more remarkable because, at that time, I was in the constant habit of performing formidable capital operations with steady presence of mind. I never afterwards went out, in the morning, without the usual *chota haziree* (little breakfast) or, when hurried, a mouthful or two of biscuit or crust. Nearly all old Indians believe, I think justly, that, to go out in the morning fasting, lays the system open to attacks of disease.

Habitual obstinate *Constipation*, giving rise to dyspeptic symptoms, occurs rarely in Europeans in India. One of my patients, for whom I used, thirty-six years ago, to have to prescribe pills which were never strong enough until croton oil was made one of their constituents, is still a healthy retired civilian in very advanced age.

Severe attacks of dysentery and of enteric fever are apt to establish habitual constipation. I believe that nine-tenths of the troublesome costiveness which I used to see early in my career, but which was decidedly less common latterly, was due to the evil practice of giving, in nearly every case of almost every disorder, from the least to the gravest, large cathartic doses from which mercury was rarely, if ever, absent. A middle-aged European gentleman, the latter years of whose Indian career I watched, killed himself, as I believe, in consequence of having become a slave to the habit of frequently taking blue pills followed by black draughts.

As I have mentioned elsewhere, I systematically refrained from giving strong purgatives in India. My usual dose was three drachms of castor oil with two of compound tincture of rhubarb in cinnamon water; or six grains of compound extract of rhubarb and two of compound extract of colocynth, with oil of cloves, in pills. To avoid danger of cholera, I never gave a purgative at night.

My able assistant at Chittagong found many of his native patients so hard to move that he frequently gave the powder of croton seeds. With East Indians, in Lower Bengal, half a tumblerful of the drastic *hot-drawn* bazar castor oil was a common dose. In many of these cases, I gave three-drachm doses which acted just as well. Commonly, however, the bowels act freely in India. In that country, I scarcely saw any of that miserable, hypochondriacal, chronic dyspepsia, with acidity and pyrosis after every meal, even of the plainest and best food, which is so common in England. This most distressing state does not occur where the skin, liver, and bowels act normally or freely, as they usually do in India. I would define English dyspepsia and 'biliousness' as chronic portal congestion, caused by habitually defective cutaneous action and constipation in a chilly climate.

For a note on *left side recumbency in dyspepsia &c.*, see page 161.

Diseases of the Intestines.

Enteritis. See *Diarrhœa*, p. 561, and *Dysentery*, p. 140.

Duodenal Ulcer.—Prep. 393 in Ewart's Catalogue is 'A portion of the duodenum which has been perforated by a lumbricus, the cephalic extremity of which is lying in the cavity of the gut, protruding as if from the finger of a glove. The external and internal margins of the aperture are even and glistening.'

Preparation E 33 in Mr. Knapp's catalogue of the Grant M. C. Museum, Bombay, is a duodenum presenting two ulcers which have perforated it. Their edges are clean cut without much thickening. The intestine was full of lumbrici; none had escaped into the peritoneal cavity. It is added—'Death was caused by narcotic poisoning.'

Dr. Chatham Gray found a perforating ulcer of the duodenum in a native havildar, *æt.* 48, who died forty-one hours after admission. The lesion was situated immediately below the pyloric ring. It was not diagnosed during life. There was no previous history of urgent symptoms.

Assistant-Surgeon O. C. Chatterjea¹ found a perforating ulcer of the duodenum in a native female.

See note of a case of perforating duodenal ulcer in a native, by Dr. T. W. Wilson, at page 549.

At pages 467 and 667 of my 'Medical Jurisprudence for India,' two cases of *Traumatic Rupture of the Duodenum* are given.

A fine well-grown young native *syce* (groom) was brought to my dispensary at Chittagong, and died rapidly from *Rupture of the Small Intestine*, a Burmese pony having kicked him in the belly. These accidents are not infrequent in the great livery stables of Calcutta.

In a Hindoo patient of mine, who died of cholera, there were found several small *Fatty Tumours* in the *Jejunum*. One of these was pendulous, ovoid, and larger than a grape.²

In a Bengali lunatic, who died of peritonitis,³ Mr. Birch found that *Round Worms* had escaped through an ulcerated opening at about one-third along the *Jejunum*.

Dr. R. D. Murray relates⁴ the case of a Burmese man, *æt.* 40, who was admitted with right pneumonia. He had two stools, vomited, and was suddenly attacked with intense abdominal pain with collapse, and died in two hours. There was recent peritonitis. A dead round worm, five inches long, lay in the peritoneal cavity. There was an aperture, about as large as a 2 anna (threepenny) piece, with clean-cut, punched-out, looking edges, in the lower third of the *Ileum*. The tissues immediately round the aperture were deeply congested. Two large round worms were found alive in the small intestine and one in the stomach. The reporter adds: 'The perforation was not the result of disease, such as ulceration; the aperture in the gut was circular and ring-like, and quite unlike any solution of continuity from disease. The mucous membrane of the alimentary canal was throughout healthy, and presented no trace of tubercular or other disease. Death, then, the reporter considered, was undoubtedly caused by the worm.'

¹ *I. M. G.* for 1882, p. 298.

² Collis and Ewart, *I. A.*, No. 21, p. 294.

³ *I. M. G.* for July 1878.

⁴ *Ibid.* for December 1876, p. 321.

Prep. E 15, in the Grant M. C. Collection, is 'A mass of enlarged mesenteric glands which formed part of a tumour weighing $1\frac{1}{4}$ lb. The ileum was ulcerated and perforated, and nine round worms were discovered in the peritoneal sac. Death from peritonitis.'

It appears questionable whether the worms perforated the intestine in either of the above cases; but evidence that round worms can perforate the tissues of the body is adduced in the chapter on *Parasitic Disease*, page 32.

In 1837, Mr. Young brought before the Medical and Physical Society of Calcutta the case of a man who died from inflammation of the ileum, caused by an immense collection (30 or 40) of round worms lodged in the intestine.¹

A few remarks upon the injurious effects frequently produced by *Intestinal Worms* will be found in the chapter on *Parasitic Disease*, pages 26 *et seq.*

Prep. 408 in Ewart's catalogue represents *Scirrhus Disease of the Ileum*. 'At the lower part of the preparation there is a cancerous mass about the size of a walnut, hollowed out in the centre by ulceration. There is a great deal of thickening and puckering of the mucous membrane in the immediate neighbourhood of the growth. Both here and higher up there are a great many smaller ulcers, and, in one place, complete perforation of all the intestinal tissues has been effected.'

Typhlitis.—An esteemed fellow-student of my own, who became Surgeon to the Governor of Bombay, died from the effects of lodgment of a gallstone in the vermiform appendix. A similar case, in England, within my experience, resulted from the presence of a cherry-stone in that situation. Prep. 75, in S. 18 of Sibthorpe's catalogue, is 'Intestinal concretions found in the vermiform appendix of an officer who died of peritonitis.' If the late Dr. Mahomed could have seen these cases, three valuable lives might have been saved.

I had to do with two cases in which there was evidence of suppuration in the cellular tissue adjacent to the cæcum, probably due to dysenteric ulceration. Both of these cases were chronic; perfect recovery took place in one of them. In both, I was surprised, considering that more or less escape of intestinal gas or of fecal matter had occurred, that sloughing was not the immediate result. Mr. John Balfour reported² an important case bearing upon this point. A military captain, *æt.* 41, of 21 years' Indian service, was subject, for two years, to chronic gastritis, irregular bowels, and discharge of blood by stool. On May 26, there was a tumour in the right iliac fossa, not moveable and with a wide base, much local pain, flatulence and rumbling. From this time, there were frequent severe attacks of local pain. The tumour gradually increased and settled down towards the groin. On

¹ *Indian Journal of Medical and Physical Science*, N. S., vol. ii. p. 347.

² *I. A.*, No. 4, p. 694.

June 10, he appeared, on the whole, in very fair health, except that the right iliac fossa was completely occupied by a tumour, generally firm, but giving a broken-up and softening sensation in the upper part. It generally felt heavy, with occasional shooting, but no permanent pain. 'So slight was the constitutional irritation that it was scarcely possible to believe that death was surely and steadily advancing.' On the 15th, the tumour had softened more below, and its contents were distinctly gaseous. Now a fœtid discharge made its appearance in the scar of an old bubo which gave way. On the 17th, sloughy shreds appeared in the wound which, on the 18th, had extended considerably upwards; a probe could now be passed three inches downwards and backwards into the fossa. The strength, which had kept up wonderfully, now began to sink. He died on the 27th. The whole of the omentum, with a portion of the transverse colon, was adherent in the right iliac fossa. In the cæcum, was a round opening with thick, almost cartilaginous edges, sufficiently large to admit the forefinger; this communicated with the iliac fossa, and, by that, with the external opening. The whole of this portion of the gut was so diseased and sloughy that it was with the greatest difficulty it could be handled at all. Only a trace of the appendix vermiformis could be found. The slough extended down to the iliac artery. There were numerous traces of healed ulcers in the transverse colon.

Dr. W. J. Moore gives¹ the case of an adult Bheel who complained of pain in the right iliac region; there was also swelling, with an indistinct sense of fluctuation. An exploring needle was used, pus issued, and, through a very deep and free incision, pus and fæces were discharged, the latter causing such intolerable fætor that the patient had to be isolated in a tent. Profuse discharge, of similar character, continued for about 19 days, when odour ceased. The wound afterwards healed quickly. The cause of ulceration did not appear.

Intestinal Concretions.

A royal lady died, in 1821, after an illness of eight days caused by obstruction of the intestinal canal by the concretion of magnesia, which she had been in the habit of taking to excess.

In the chapter on *Diseases of Pregnancy*, I have noticed the habit, which is very general among Bengali women, of eating baked clay cups. In a case where a woman usually indulged in these when pregnant, a rounded mass, about the size of a walnut, was passed from the bowels, and was sent to the Calcutta Medical Society by Dr. Greene, and Baboo A. C. Sen of Tipperah. The patient had been subject for fourteen years to severe pain in the stomach, with incessant vomiting and tenderness on pressure in the epigastrium. Dr. McConnell found

that the nucleus was formed by a small betel nut. The crust is thick, very dense and hard, and appears to consist purely of phosphate and carbonate of lime. The clay-eating was probably only coincidental. Two other intestinal concretions were also brought for exhibition by Dr. MacConnell. Prep. 1658 in the M. C. museum, was removed by Dr. Matthew from the rectum of a man at Midnapore. After symptoms of intestinal obstruction and pain and weight in the rectum, the calculus (size and weight not given) was removed by the finger and a scoop. It is described as being laminated, and principally made up of oxalate of lime, with cystine in addition. The nucleus appeared to consist of vegetable fibre in a state of fine subdivision. It seems probable that the analyst's notes had become mixed. Prep. 2279, presented by Dr. MacConnell, a concretion of the size of a small orange, was removed from the rectum of a native boy of 10 who had suffered for ten days from bowel obstruction. It consists, almost exclusively, of the husks of some graminaceous plant.

DIARRHŒA.

The S. C. R. show that, in 1881, the deaths, in Jails, from dysentery and diarrhœa were per mille to strength:—Bengal, 17·91; Lower Bengal and Assam, 33·74; Madras, 20·62; and Bombay, 12·01.

The total deaths, in the three Presidencies, during that year, were, from dysentery, 987, and from diarrhœa, 728, in a jail population of an average strength of 100,844, these two being the diseases from which the largest mortality resulted during the year.

In 1883, the admissions, for diarrhœa, of European soldiers, amounted to 2,548 with only 2 deaths; 2,443 native soldiers were treated, of whom 43 died. The admissions of prisoners were 7,504, and, of these, 483 died.

Hence it is to be inferred that, while diarrhœa is common but comparatively harmless among European troops, it is a somewhat formidable disease in the native army [in 1881, there were 151 deaths in the three Presidencies, this disease being fourth in destructiveness in that year] and that it is one of the deadliest maladies to which the poor of India are subject—the deaths in jail being generally an approximative measure of the health of the general population in any given locality. This test is, however, least applicable in regard to diseases of the alimentary canal, seeing that the prisoners are better fed than their outside brethren are.

I must repeat that, in treating bowel complaints, it is to be borne in mind that the rice-eater of Lower Bengal has, as a general rule, two stools in the twenty-four hours.

In a very large proportion of cases, especially of those in which the flux is transitory, *diarrhœa is an expression of hepatic disorder* which

is generally functional, but which may be organic. Most cathartics exert a direct or a reflex cholagogue action upon the liver. A debauch frequently gives rise to a copious flow of bile and loose stools. Most strangers, before their first month's stay in Paris is over, get an attack of diarrhœa from eating rich made dishes to which they are not accustomed. In the white chronic diarrhœa of the tropics, the absence of bile pigment in the stools, often for months, nay years, appears to point to hepatic lesion. Cases of liver abscess often terminate fatally by diarrhœa, whether there has or has not been previous dysentery. Therefore, for many years before I left India, my treatment, in obstinate cases of diarrhœa, was mainly directed to the liver.

In India, *Crapulous Diarrhœa* occurs nearly as it does in England, save that, in the two countries, the causes often differ. Thus, in India, the inexperienced practitioner may be surprised at finding that the evacuations consist mainly of the large seeds of the guava or of horse beans (*gram*). Sherbet made from the ripe bael fruit (*Ægle marmelos*) is a favourite laxative with many in India. The fruity matter seems to pass through the intestinal canal almost unchanged, having, in the evacuations, much the appearance of scraped apple. A brother officer of mine took a tumblerful of this mixture early one chilly morning in the rains. He was not in strong health at the time, and swallowing this large quantity of cold indigestible matter caused a shock to the ganglionic nervous system almost as severe as a blow in epigastrio would have done. For a little time, he was almost collapsed. A healthy European, having business in Calcutta which caused him to knock about a great deal on a very hot day, drank repeatedly large quantities of iced water. The result was an attack of diarrhœa.

What would probably, if properly treated, be merely the diarrhœa of a few hours, is not infrequently much aggravated, nay, perhaps rendered chronic, by imprudent exposure to vicissitudes of temperature, and by erroneous medication. A queen's officer, holding the position of P.M.O., consulted me for an obstinate diarrhœa which had troubled him for some time. He looked very ill, and I feared that the case would prove obstinate. He was passing light-coloured thin stools, and was taking frequent small doses of hydrargyrum c. cretâ. I entreated him to drop the mercurial. He did so, and was almost immediately well. It is here noteworthy that mercury with chalk (which is, I believe, in England, only noxious as a mercurial) is a poison in India. If an ordinary dose be given to a child, the little one turns pale in a few minutes and throws it up. It has been shown by Drs. Burton Browne and F. Macnamara that, in India, grey powder, which has been kept, contains from 4 to 20 per cent. of the peroxide (red oxide) of mercury.¹ I cannot but think that long keeping must give rise to a like change in a temperate climate. If I am satisfied of any fact in

¹ *I. M. G.*, vol. i. p. 205,

therapeutics it is of the truth that the use of grey powder is not indicated in diarrhœa, or in any other disease.

Infantile Diarrhœa (*vide* p. 534).—In Bengal, an infant rarely goes through the process of teething without suffering more or less from either diarrhœa, coryza, cough, dysentery, or the whole of these complications. In 1883, the European soldiers' children in all India had 524 admissions for diarrhœa (87·1 per mille of strength), with 51 deaths (8·48 per mille of strength). As a general rule, the incisors are cut easily, and, up to the age of twelve months, the child is well grown and intelligent. Then, for from four to eight months, until the canine teeth have been fully cut, there is a period of grave ill-health, usually from bowel complaint, the child becoming languid, of dull intellect, and emaciated. Then, at and after the age of two years, another, but usually less severe, time of trial comes, during the cutting of the molars. In the native and East Indian child, the commencement of dentition is often late. Here, especially when the hygienic surroundings of the infant are bad, the process of cutting both incisors and canines nearly at the same time becomes a great and very dangerous trial. Much of the time of Indian medical men is occupied in attendance upon these cases. Potentially, we can do little except to see that the nurse is adequate, and to lance the gums sufficiently *at the proper time*—that is to say, knowing precisely what teeth are coming, and at what time each is likely to be due. We do not, however ill the child may be, incise the pale gum over a tooth which is not pressing forward; but we must never fail to lance, in illness, when a tooth is due and the gum swollen and vascular. I steadily followed this practice throughout my Indian career, although I found that some mothers and also some medical men were strongly averse to it; but not one of the objections alleged against it had the very slightest weight in my mind. Our collector, at Chittagong, was a rather noted and very inveterate amateur in medicine. I lanced his child's molars, which bled a little freely, in his presence. He looked at the child, and then ran up to me, wringing his hands and crying, 'There's hæmorrhagy!' The reply was, 'It will do good, and cease almost immediately.' A little neatness of arrangement much facilitates this minor operation. The child being in the nurse's arms in the next room, I placed two chairs, facing one another, near the window, so that the full light would fall upon my chair. The mother would seat herself facing me. The nurse would bring the child and place it, with its back towards me, on the lap of its mother, who would, engaging its attention, allow its head to fall gently backwards upon a pillow which crossed my knees. There would then usually be an attempt to cry, and I made a St. Andrew's cross through each swollen gum, *feeling the tooth all the way*. Then the mother ran off with the child. In this way, the infant was neither pained nor frightened, and never appeared to bear me ill-will. It very rarely happens that the bowel com-

plaint kills these children, although, attempt to soothe it as we may, *it will continue until the teeth are cut*, and often much longer, so that change of air to England in the case of European children will be needful. Hence, in a large measure, the pallor and weediness of English children in Bengal.

Nearly every well-found ship, returning to England from India, contains miserably emaciated children with diarrhœa or dysentery. In my three voyages home, I never saw a death. As a general rule, although wretchedly weak and fractious, these poor infants improve greatly on the voyage. In Bengal, there is probably a large mortality among native children in teething; but, among well-to-do people, I only saw two deaths from infantile diarrhœa—in an English child and in one of mixed blood. Generally, the European woman in India is a most exemplary mother. I never saw or heard of a case in which one failed to nurse her infant, save on compulsion. A healthy native wet-nurse (dhye), knowing her own value, is apt to be ill-tempered and tyrannical, is often gluttonous, and, unless very closely watched, is likely to drug the child with opium to save herself trouble by keeping it quiet. Still, these women are often useful. Where the bottle was employed, I generally recommended the milk of a grain-fed goat. Two kinds of goat are preferred: a very large, tall, stag-like animal—*Jumnapari*—and a long-bodied goat with short strong legs—*Bengali* goat.

I have spoken, under the head of Diseases of the Liver, p. 606, of the transient white diarrhœa to which children of India are liable when exposed to the chill of the early morning. The use of a flannel coverlet, drawn up before daybreak, and keeping the children at play indoors on damp mornings, would often prevent these attacks.

Besides the acute *Bilious Flux* of which notice is taken in the chapter on Cholera (page 296), there is a more or less chronic *Bilious Diarrhœa*. It may end in dysentery or hepatic abscess. Its treatment is by free counter-irritation over the whole hepatic area, a course of small doses of ipecacuanha, plain food in great moderation, rest, and, if possible, a sea-voyage.

As I have shown at the commencement of this chapter, diarrhœa is the most destructive malady prevalent in the jails of India. In 1883, there were 7,504 admissions, with 483 deaths. In Bengal Proper and Assam, 3,138 admissions, and 101 deaths; Chota Nagpore and the Gangetic Provinces, 1,007 admissions, 65 deaths; Central Provinces, 424 admissions, and 114 deaths; Agra and Central India, 165 admissions, and 15 deaths; Meerut and Rohilcund, 210 admissions, and 12 deaths; Punjaub, 539 admissions, and 47 deaths; Bombay Presidency, 474 admissions, 38 deaths; Berar, 24 admissions, and 2 deaths; Madras Presidency, 603 admissions, and 64 deaths; British Burmah, 461 admissions, and 9 deaths; Andamans and Nicobars, 459 admissions, and 16 deaths.

During this year, the admissions per mille of strength were: All India, 85·0; Bengal Presidency, 99·6; Lower Bengal and Assam, 252·5; Chota Nagpore, Dinapore, Benares, Oudh, and Cawnpore districts, 59·6; Central Provinces, 162·5; Agra and Central India, 37·9; Rohilcund and Meerut, 31·7; Punjaub, 44·5; Bombay Presidency, 60·5; Berar, 23·6; Madras Presidency, 78·7; British Burmah, 89·7; Andamans and Nicobars, 39·9.

As a general rule, Diarrhœa and Dysentery (vide p. 141, for statistics of jail dysentery) are nearly equally prevalent and destructive in Indian jails.

A glance at the above rates must lead to the observation that, in the jails of Lower Bengal and Assam, where the staple food of the poor is bad new rice, the admissions and deaths under the heading 'diarrhœa' vastly exceed the rates from the same disease in every other part of India, where, as a general rule, the poor can obtain a better staple of food, and are, consequently, for the most part, healthier and of better physical development.

Here we must also take into account the greater prevalence and intensity of malaria in Lower Bengal.

A good deal of attention has been paid to the fact that, especially in this Province, the prisoners and the agricultural poor suffer thus grievously from destructive intestinal disease, but this extremely important subject has never received the systematic investigation which it demands. I trust that the following observations, the outcome of painfully large experience, and of much anxious thought, may suggest the necessity for an exhaustive inquiry.

MORBUS BENGALENSIS.

In Indian diarrhœa, the causative influence of malaria must never be lost sight of.

This subject is prefaced by my remarks on Indian Dietetics, p. 23.

In 1826, Mr. J. Tytler submitted to the Medical and Physical Society of Bengal,¹ a paper in which this great question was fairly opened. He argued that three-fourths of the total deaths among the lower orders of the natives of Hindostan are due to diarrhœa, the malady either occurring idiopathically or as a terminating symptom. 'It is,' he contends, 'the scourge of crowded jails and of hospitals filled with harassed and fatigued sipahies, and is the great avenue through which the swarms of naked and famished paupers and miserable infants that occupy the native bazars are continually vanishing.' Tytler designated this condition *Diarrhœa Hectica*. I however found that, in a very large proportion of these cases, as they occur in Lower Bengal, dysenteric symptoms present themselves from time to time

¹ *Transactions*, vol. iii. p. 1.

during the term of the patient's decline. It therefore appeared to me that—taking also into account the fact that this is the most prevalent form of disease in Lower Bengal—the malady may be fitly designated as 'Morbus Bengalensis.' The general history of the many hundred persons who died of this cachexia in the beds of my hospitals—I have no recoveries from the confirmed disease to boast of—was clear and very uniform. They were rice-eaters of the poorest classes. They were mostly adults. They came under medical treatment in an almost extreme condition of emaciation. They were the subjects of chronic diarrhœa, often alternating with dysentery. When there was simple diarrhœa, the stools were merely loose and frequent. The dysentery was not usually very severe or attended with sloughing. They generally appeared to improve, under treatment and good feeding, for a few weeks. In many cases, *the bowel complaint was arrested*, and, consequently, vain hope of recovery began to be entertained; but, after a month or five weeks, emaciation and debility made steady progress and, usually within two months, the patients sank—to all appearance starved to death—perhaps less rapidly, but as certainly as if this complication had remained unchecked. I used to tell my students that Morbus Bengalensis is no more diarrhœa or dysentery than a dragoon is part of his horse. True, they are usually seen together, but they are distinctly separable and very dissimilar entities. In many p. m. examinations, I could find no lesion of mesenteric glands, and I could discover no marked or characteristic traces of disease in the intestinal canal, save that, now and then, but not at all invariably, there were traces of moderate ulceration in the large intestine. I saw nothing of this condition among well-fed natives, Eurasians or Europeans, but, now and then, it occurred in my practice among natives of rank and wealth of the old school, with whom it was deemed orthodox to undergo semi-starvation in advanced age and failing health. Men suffer far more from this condition than women, which is probably due to the fact that, hard-worked and ill-fed as the women are, the men work harder upon an equally inadequate diet.

After I had treated these cases hopelessly for years in district jails, I began to make inquiries regarding this malady as it occurs in and around Calcutta. It was suggested to me by my friend, the late Dr. John Brown, that he had found it possible to arrest the process of tissue waste in these cases by allowing a generous diet. I tried this fully, in every imaginable way, for years. At that time, medical officers in civil employ were not restrained by the Government in dieting or drugging the sick poor. Consequently, for years, I prescribed each patient's daily food, giving wheat flour (soojee, attah, bread), eggs, strong kid soup, milk, curd (duhee), ghee, dhall (lentils), eggs, wine, old rum,¹

¹ New rum is always poisonous from the presence of fusel oil. I never gave any but old and well-seasoned spirit.

the best rice, sugar, and, seeing that a scorbutic taint probably existed, anti-scorbutic vegetables and fruit. It may be thought that I now erred by allowing too free a dietary to persons unaccustomed to good food. I was always prepared to avoid this error; and, when the patients were not absolutely moribund on admission, I almost invariably found that they, for a short time, distinctly benefited from the food which I gave them. I adopted, in these cases, the system recommended by Meer Ushraff Ali, the late estimable teacher of midwifery, of thoroughly rubbing some ounces of ghee (buffalo butter), or mustard oil into the whole body daily, the patient lying naked in moderate sunshine. This always did marked but transient good. If I had power in India, I would appoint an experienced medical officer to make a special study of this morbid condition, and of the class of maladies known as *Diarrhœa Alba*. He should have a small hospital, well constructed upon the principle of a native hut, but with none of the deficiencies and sanitary faults of a native dwelling; and a dead-house, in which work can be done, like the little building with walls of wire-net which Dr. Payne contrived for the native lunatic asylum at Calcutta. Annexed to these should be a laboratory and a microscope room. The work of the cook-room and the distribution of each meal should be personally seen to by trustworthy medical subordinates. Into this hospital, as many patients suffering from diarrhœa as the medical superintendent might, from time to time, call for, should be sent, in covered and well-fitted ambulances, from the nearest jails and hospitals. The history of every patient, especially as regards his mode of feeding, should be carefully taken. Every stool should be examined. The condition of the entire length of the alimentary canal, epithelium, villi, lacteals, mesenteric glands, follicular apparatus, ought to be exhaustively investigated; due attention being paid to the not very extensive but most important literature of the chronic diarrhœas of tropical and sub-tropical regions, of which I am about to give a necessarily concise outline, and especially to the pathological details given in Dr. D. D. Cunningham's account of the diarrhœa and dysentery of the famine districts of India published in the Fourteenth Annual Report of the Sanitary Commissioner for 1877. The idea which these cases first suggest is gradual starvation from arrest of nutrition in the lacteal system. But the stools are not bulky, as in English mesenteric disease, and, as I have already said, I was unable to detect enlargement or disease of the mesenteric glands. The dietary should only be limited by the physician's discretion. I believe that, well and thoroughly carried out, an investigation of this kind might be, potentially, the greatest boon that England could bestow upon India. Still, my own sad experience leads me to fear that this evil lies beyond the reach of medicine.

The late Mr. Strong, surgeon to the 24 Pergunnahs (Calcutta),

devoted his spare time, during a very long career, to the duty of showing that the great curse of Lower Bengal is the use of new *dasee* (common) rice, as the staple diet. Assuredly the commonest rice is always unwholesome, especially when it has not been stored for at least a year, but it scarcely comes within the power of the wealthiest government to improve the food staple of a population of millions. In treating these cases, I found that the Bengali is never willing to relinquish a daily meal of rice with *dhall* (lentils with oil and red pepper), but he is almost always glad that his second meal shall principally consist of wheat meal. I believe that, if this system could be introduced in every hut throughout Lower Bengal, the strength and health of the Bengali race would be enhanced; but, unlike the English labourer, the native of India scarcely recognises the existence of luxuries in food (beyond a copper's worth of sweetmeats, for which the condemned criminal invariably begs), and habitually deprives himself of a due quantity of even the scanty innutritious food which he is able to command, as a measure of economy or as a religious duty. In February 1870, as President of the Bengal Social Science Association, I used the following expressions at our annual meeting: 'As the most usual and natural cause of death among the English poor' [at home] 'is some form of chest complaint, so the Bengali labourer generally falls a victim, early in life, to a malady described many years ago by Dr. Tytler as *Diarrhœa Hectica*, but which, on account of its universal prevalence, I would name *Morbus Bengalensis*. I find, in Dr. Mouat's tables, that, out of 17,380 deaths from all causes which occurred in the jails of Bengal in the decennial period 1857, 1865-66, no less than 8,024 were from dysentery and diarrhœa. No system of jail sanitation will prevent this. . . . Having watched this disease closely for more than twenty years in an extensive field, I am perfectly convinced that the *Diarrhœa Hectica*, although it is its usual concomitant, is no essential part of the malady, which is a gradual failure of the powers of digestion and nutrition. It was long hoped by many of my professional brethren and by myself, that this disease would yield under a very generous diet. I have now, however, for nine years, tried upon these patients the effects of every imaginable mode of feeding, and I find that no form of diet that we can devise ever produces more than very temporary benefit—the patients dying, physiologically, in a state of starvation, however well they may be fed. I repeat that my distinct conviction is that this inability of the system to receive nourishment is mainly due to the slow failure of the nutritive faculty under a lifelong system of feeding upon poor, bad, and indigestible food. I believe that a state of famine which, although almost imperceptible to the ordinary observer, is, on that account, more widely, stealthily, and surely destructive, constantly prevails in Bengal. Born of a famine-

stricken race, nursed by a starved mother, working hard upon a food the use of which is slow starvation, the Bengali's life burns out rapidly, as the feeble light does in his poor *chiragh*, and for the same reason—want of fuel. I believe that I am not original, when I say that rice has ruined Bengal as the potato has ruined Ireland. Both countries are endowed by nature with soils of great fertility. In the middle ages, Ireland was celebrated for her herds of cattle, which she exported abundantly to England and other countries. Bengal was, at about the same time, a land of great pastures, upon the rich grass of which the sacred herds fed in such multitudes that they were offered by ten thousands in sacrifices to Kali. I consider that the only valid remedy for the *Morbus Bengalensis*, as well as the only means of enabling the Bengali to withstand the marsh poison of his country, until he shall have driven out the malaria by bringing his rice-swamps into dry cultivation, is *the introduction into Bengal of a higher staple of food*; and I would earnestly press upon the Association the necessity of full inquiry and mature deliberation upon the questions: (1) how far it may be possible to widen the pasturage of Bengal and to extend the cultivation of wheat and legumes, of oil-producing plants, and of fruit trees, especially the mango, throughout the country; and (2) to what extent, in the mean time, the opening of ports along our sea-board, which now engages so much attention, and the spread of railways through the most fertile tracts of India, may enable us to import wheat into Bengal until the country can import its own wheat. It will give me great satisfaction to find that the important question which I have now mooted is considered worthy of being dealt with by the *Health Section*.'

It is to be feared that my remedy for *Morbus Bengalensis*, although sound in theory, is practically Utopian. An experiment of this kind is not to be expected of the zemindars of India, and the poorest Bengalis will never take to wheat meal, as a staple of diet, until it can be obtained as cheaply as dasee rice.

Tytler's description of the symptoms and progress of this disease is admirably graphic and true. He hoped much from the use of opium and tonics; but they proved as uncertain in his hands as in those of his successors. Indeed, diarrhœa is one of the most usual modes of death to which the opium-eater is liable. An account of this cachexia, as occurring in the jail of Coimbatore, Madras Presidency, is given¹ by Dr. Geo. Mackay. In most of his cases, there was so much disease of the large intestine that, had they occurred in my practice, I should have returned them as cases of dysentery.

True *Morbus Bengalensis* is not characterised by extensive ulcera-

¹ *I. A.*, No. vi. p. 549.

tion ; but, of course, Morbus Bengalensis does not afford any exemption from dysentery. Out of twelve cases in which Dr. Mackay examined the liver microscopically, six showed the tissue of the liver and hepatic cells to be loaded with fat globules, a fact which the author considered to be of considerable importance with reference to the pathology of the disease. But it is to be borne in mind that these are cases of rapid and extreme wasting.

Writing from Gyah in 1830, Mr. James Hutchinson gave an interesting review of this class of cases.¹ He placed considerable reliance upon three-grain doses of ipecacuanha with opium, and upon a diet of the best rice mixed with curds (duhee).

Dr. Kinloch W. Kirk, an excellent observer, wrote in 1844 upon this disease² as it then occurred in jails and in native regiments. He contended that, 'if ever a cognomen did harm, the name "Chronic Dysentery," applied to this affection, had done so by communicating an improper idea of the nature of the disorder, and, consequently, giving rise to an erroneous treatment. The disease is essentially colliquative diarrhœa arising in the course of low typhoid' [i.e. typhus-like] 'fever, though sometimes a dysenteric character is produced by the following accidental causes: (1) Exposure to cold in the night, especially if the patient sleep on the ground. (2) Swallowing an excessive quantity of half-cooked food. (3) Eating too freely hot spices, when not in the habit of using them. One or more of these causes may generally be found to have operated in jails; the second is that which obtains most frequently in the hospitals of the native regiments.' Dr. Kirk relied much upon opium and quinine, and strongly objected to mercury and ipecacuanha. He recommended small enemata containing ten-grain doses of acetate of lead, which was at that time a drug much employed in alvine fluxes. I did without this poison, both in bowel complaints and in hæmoptysis, for many years before I left India.

Chronic Tropical Diarrhœa (Diarrhœa Alba) in Europeans.—Writing in 1829, Twining observed³ that, when dysentery has been protracted and assumed the chronic form, certain patients suffer from the continuance of a copious watery purging, sometimes attended with the appearance of fermentation like a mixture of chalk and beer; often a portion of the aliment passing undigested, and the fæces being of various colours, but mostly of pale grey, and slime or a white matter abounding, while blood was rarely present in the stools. For this,

¹ *Trans. of the Medical and Physical Society of Calcutta*, vol. v. p. 39, 'On the Alvine Fluxes of the Natives.'

² Of the disease called Chronic Dysentery, frequent among prisoners, *Ibid.* vol. ix., part i., p. 217.

³ 'On the Employment of Large Doses of Ipecacuanha in Dysentery,' *C. M. T.*, vol. iv. p. 198.

Twining gave ipecacuanha, and applied a blister, kept open for many days, to the belly. Except in scorbutic cases, Twining restricted the diet to bread and tea, or milk and sago—not more than six ounces of food and drink together being taken at one time. He added soups in limited quantity during the progress of recovery, solid food of any sort, except bread, not being allowed until convalescence was far advanced. Bleeker described this disease, attributing it to diminished power of absorption in the large intestine consequent upon the destruction of mucous membrane by dysenteric ulceration. He prescribes a carefully regulated diet, morphia, and astringent decoctions. He adds, ‘In such cases, nitras argenti fusum, internally and in injections, generally produces good effect. I have given this preparation internally in increasing doses, from one grain to a scruple during the day, and I count with pleasure numerous cures without having to regret any unfavourable effects.’¹

If this chronic white flux be an outcome of dysentery, it is singular that it never followed that disease in any of the many hundred cases which came under my care in Bengal. To say that these patients have suffered from dysentery is merely to say what may be said of nearly everyone who has resided in India. Morehead² considered it ‘an error to suppose, as many do, that this diarrhoea is symptomatic of hepatic derangement. No doubt the secretion of bile is deficient; but can it be otherwise when the system is anæmic, and an active derivation of the fluids to the intestinal surface is going on?’ It is self-evidently a matter of the greatest practical importance that we should know more than we do at present of the conditions of the liver in *diarrhoea alba* uncomplicated with jaundice. If Morehead’s opinion be correct, as it probably is, nothing can be more futile than to treat the disease as one of hepatic origin. We ought to ascertain with precision to what extent bile-elements are present in the yeast-like evacuations. We must not trust too much to colour—to the *apparent* absence of bile-pigment in the dejecta which, in health, do not assume the natural brown appearance until they are received into the large intestine. Should an officer be appointed to make special inquiry into *Morbus Bengalensis* and *Diarrhoea Alba*, one of his leading objects would be to make a thorough examination of the urine in these cases; as this would probably lead to facts showing whether the kidneys take any part in eliminating the bile elements in which the stools are deficient.

Alexander Grant³ and Dr. P. Mason⁴ describe, the former having experienced it in his own person, a *Diarrhoea Alba* known in China and Java.

¹ *Op. cit.* p. 27.

² *Op. cit.* p. 317.

³ ‘On Hill Diarrhoea and Dysentery,’ *I. A.*, part i., p. 324.

⁴ *Fayrer’s Lettsomian Lectures*, p. 143.

That we may take a large view of this class of maladies, it will be well to consider here the subject of *Hill Diarrhœa*, an admirably exhaustive account of which was given by my friend, Mr. Alexander Grant, in 1854.¹ Here, it is to be premised that, in ascending the Himalayas from the plains, it is generally noticed that the change of temperature gives rise to rather troublesome diuresis. This result of checked cutaneous action is also experienced, often for some months, by old Indians on their return home—in these latter, there is frequently a considerable tendency to diarrhœa, the stools being deficient in bile-pigment. This may occur, from time to time, during the earlier years of residence at home, especially when the weather suddenly becomes cold and damp. Most certainly, diarrhœa alba is not a disease either of the European or the native inhabitants of India, as long as they continue to dwell in the plains. Morbus Bengalensis, a disease of the natives of the plains of India, is a malady perfectly distinct from this *diarrhœa alba* of Europeans, although it resembles it in various points—most especially in its incurability when treatment is long deferred. Grant shows that few persons escape one or more attacks of the diarrhœa, known by the slang phrase of ‘Simla trots,’ in the course of the season. Sir Ranald Martin says, ‘the returning Indian is often seized with diarrhœa from exposure to cold on board ship, especially if exposed to the spring easterly winds.’ Fayrer largely illustrates this point. I have certainly seen more of diarrhœa alba in persons returned from India than I ever saw in India. Grant shows that the ‘trots’ may merge in true and inveterate *hill diarrhœa alba*. The attacks, he observes, are not confined to persons in broken-down health, who have suffered from periodic fever or other tropical diseases; on the contrary, residents in the prime of life and of sound constitution, have been very generally affected, and, among these, examples of its proving fatal are not rare. The commencement of the rainy season, when the air, from being dry and warm, becomes moist and cool, is the period of greatest prevalence of this disease. Among the predisposing causes, Grant gives a prominent place to scorbutic cachexia and to the strumous habit, previously impaired health, the taint of marsh poison, and the imprudent personal habits of soldiers—‘the temperate, however, suffer equally with the intemperate; but the mortality among these last is much higher.’ Grant considers that bad conservancy, at the old hill stations, was largely operative in the causation of this malady, and I recollect that he foretold the occurrence of hill diarrhœa at other more recently occupied hill stations should proper conservancy arrangements not be provided there. This came to pass, both at Mount Aboo and in the Neilgherries. The process of slow exhaustive decay of body and mind under a process of steady drainage by frequent loose, frothy, or yeasty stools, in the early stage quite white, later of a

¹ *I. A.*, vol. i. p. 311.

pale drab or muddy grey colour, afterwards pultaceous in consistence, is admirably described. Grant attributes the condition of the stools to a 'depraved and diminished secretion of bile.' 'In all cases,' he says, 'there is arrest of the biliary secretion, and it may be pronounced that the disordered function of the liver is an essential characteristic of the disease. Relapse is common and easily induced, as by an error in diet or by a change of weather, as from dry to wet.' Ardent spirits act as a poison.

I think that the general consensus of observation points to the fact that, when not relieved early, this disease has a very strong tendency to destroy life. With regard to the pathological condition of the bowels, Grant observes that, 'In some cases of wasting painless diarrhœa, there are no organic lesions to account for death; the bowels, like the body, are pale, blanched, and anæmic, or the coats of the intestines are strikingly atrophied, and in places diaphanous. This applies peculiarly to the small intestines, while there are leaden spots in the colon, which is easily lacerated.

'In other cases, the mesenteric glands and the mucous follicles of the intestines are enlarged, and the calibre of the gut is narrowed in places, and the mucous membrane softened, pulpy, and hypertrophied, with serous exudation under it, or simply thickened, and in a red granular state.' E. Goodeve notices¹ the wasted condition of intestine described above. 'In some chronic diarrhœas, especially those with the white flux, in spite of the long duration of the disease, little or no change of the mucous surface is to be found. There is great thinning of all the coats of the small intestines, so that they are quite translucent, and there is doubtless atrophy of the glandular tissues.' Goodeve describes intestinal amyloid degeneration. Dr. Aitken observes that lardaceous disease of the intestines, nay, of the whole tract from mouth to anus, is frequent in certain forms of diarrhœa. It involves the arterial capillaries of the villi and surrounding net-works of mucous and sub-mucous tissues; it progressively involves the villi, mucous and sub-mucous capillaries, infiltration around the solitary glands, and degeneration of the vessels surrounding the Peyer's glands.

In a paper read at the International Congress at Copenhagen in August 1884,² Sir Joseph Fayrer and Dr. Joseph Ewart observe that bill diarrhœa is essentially periodic, it is marked by progressive deterioration of the secreting apparatus with a tendency to shedding of the epithelium of the villi. Granular and fatty degeneration are generally present and may be accompanied by amyloid infiltration. The liver and kidneys are often found in a state of chronic inflammation.

¹ Art. Diarrhœa, Reynolds's *System of Medicine*.

² 'On Chronic Diarrhœa and Dysentery in Persons Returned from Tropical to Temperate Climates,' cited *M. T.*, vol. ii. for 1884, p. 264.

Grant observes that, in these cases, abscess of the liver is rare. In general, he found this organ small, pale and friable, especially in the anæmic cases.

An aphthous condition of the mucous membrane of the mouth, which probably extends throughout the whole of the gastro-intestinal tract, is frequently observed in these chronic diarrhœas.

Treatment.—*The maintenance of strict recumbency is indispensable in all obstinate cases of Diarrhœa.* Grant urges the importance of means tending to speedy restoration of the biliary and cuticular secretions, by moderate cholagogues and confinement within doors. In obstinate cases, he trusts almost solely to a combination of aromatics and astringents concurrent with a strict attention to diet and clothing, measures tending to promote and rectify the secretion of the liver, and the patient's removal to a less humid and non-malarious climate, to Cashmere or to Europe. All Indian experience confirms Mr. Grant's recommendation that the flannel cholera belt should be constantly worn.

Mr. Grant has lately informed me that he continues to hold the above opinions, as expressed in 1854. He considers that, if advance has been made in the treatment of this class of disease, it is in the direction of diet, and not in the efficacy of any particular drug. In a recent case of great obstinacy, Mr. Grant called attention to a paper 'On some new articles of Peptonised Food prepared by the Pancreatic Method,' by Dr. W. Roberts, of Manchester; p. 517, vol. i. of the 'Transactions of the International Congress' of 1881. The directions therein laid down were carried out to the letter. Recovery was complete.

I have little doubt that at least some of the many kinds of peptonised foods, which are so much advertised in the present day, have been employed in the chronic diarrhœas of India, but I have not met with reports of such treatment. One of the results of my own narrow observation upon this point is that there is great difficulty in finding compounds of this class which are relished by patients. It is quite useless to attempt to press upon these unfortunates any food, however self-digestible it may be, if they consider it unpalatable and regard it with disgust; still I think that the employment of the best of these foods deserves fair trial, providing this difficulty can be overcome.

In treating these cases, it is to be borne in mind that, unhappily, during the months or years occupied by the advancing course of this disease, the appetite is so keen or rather so voracious, that, as also happens in most of the worst cases of sub-acute dysentery, the patient is almost to be regarded as a monomaniac on the subject of food. It really appears as if, *Diabolo suadente*, his leading instinct, nay, almost the sole care of his life, is to satisfy an unnatural greed for all those articles of diet which his own common sense ought to warn him are most unsuitable to his condition. At the same time, there is

generally here, as in cholera or sub-acute dysentery, a great loss of digestive power which usually enables us to detect irritating masses of food in the motions. Patients of low morale will steal and lie in satisfying and concealing this morbid craving; and the best and otherwise most sensible men and women are generally very unmanageable and disobedient in this respect. Mention has already been made of the value of curds as a nutriment in this class of diseases. Fayrer relies upon milk alone or diluted with about one-fourth or one-third part of lime-water, also beef tea, raw beef-juice, or other plain animal broth 'free from extraneous matters.' To every one who has to treat cases of this kind in Europe, Fayrer's chapter on treatment in his third Lettsomian Lecture, page 144, is full of invaluable teaching. As laid down in the paper read before the Copenhagen Congress, the line of treatment enjoined by this authority is 'Absolute rest, good ventilation, and purely milk-diet, given at first in small quantities and frequently and gradually increasing in quantity until from three to four quarts are consumed in twenty-four hours. Occasionally, small doses of opium may be given, but other drugs are to be avoided. The time for gradual return to ordinary diet must be determined by the circumstances of each individual case.'

Dr. W. P. Dickson¹ attributes hill diarrhœa mainly to impure water supply. Consequently, he recommends that drinking water should be boiled or filtered. To this we must all agree, because, everywhere in India, the first law of domestic hygiene is that every drop of drinking water must be filtered and boiled. This is another argument in favour of treatment by pure milk.

Important contributions to our knowledge of Hill Diarrhœa have been made by Drs. Green² and Moore.³

Famine Diarrhœa.

The organic lesions by which dissolution is brought about in cases of starvation can hardly be recognised as disease. Still a good many facts of pathological importance have been learnt by observers of Indian famines.

My lamented friend, Dr. Lewis, of Netley, kindly obtained for me the following data, from the official returns, with regard to the great famine in Madras in 1877. In 1875, the population of the Madras Presidency was 29,299,586. In the six years 1875-80, excluding 1877, the death-rates from all causes, per 10,000, were 211, 233, . . . 278, 189, 157; in the famine year, 1877, they rose to 532.

¹ *I. M. G.*, vol. vi. p. 47. 'The Cause of Hill Diarrhœa Exemplified by the Sickness from the Disease in the Dhurmsala Jail.'

² *I. A.*, No. 2, p. 517,

³ *Bombay Med. and Phys. Trans.*, for 1869, p. 109,

Some relics of this terrible national visitation are found in the museum of the Medical College at Madras.¹

134. Part of ileum and colon. Taken from an old man who had been ill fifteen days with 'famine diarrhœa.' The former shows congestion, slight thickening with aphthoid exudation on its surface; the latter showing sphacelus of the whole of the coats and sloughing ulcers in parts, whilst, in other places, the tissue is quite healthy. He was under treatment during the last ten days of his illness, had no griping or tenesmus; belly soft; pain on pressure near navel, stools thin, mud coloured or greenish or mixed with venous blood. No feverishness; died from exhaustion.

135. Taken from the body of a native female, *æt.* 35, who died from 'famine diarrhœa' after an illness of five weeks. Distension by pus of Peyer's glands, surrounded by infiltration. Some have discharged their contents, leaving circular ulcers without infiltration. Similar ulceration is seen in the sigmoid flexure. There is no vascularity of these parts. The jejunal mucous membrane is pink and swollen, and there is punctated ecchymosis of two of Peyer's patches; there is also an ecchymosed spot, the size of a split pea, on the jejunum.

In both of these cases, in that of the man especially, we perceive the evidences of scorbutic action.

20. Atrophied spleen, weighing only 5 drachms. Taken from the body of a cooly, *æt.* 55, height 5 feet 5 inches, weight 83 pounds, who, during the famine, had been ill with diarrhœa for a month when pneumonia supervened, from which he died in a week.

22. Spleen weighing only half an ounce. Taken from the body of a female, *æt.* 50, who died from 'famine diarrhœa,' from which she said she had been ailing for only four days. The body was much emaciated, weighing only 55 pounds. There was sloughing ulceration of the colon. The whole of these specimens were given by Dr. A. Porter. They all point to the necessity for the free use of antiscorbutic treatment in famine hospitals.

Opium Eater's and Smoker's Diarrhœa.—All who have seen much of these unfortunates recognise the fact that, in India and China, those habituated to the use of opium are very liable to fall victims to diarrhœa. The following remarks, in the old 'Madras Topographical Reports,'² still hold good. 'Some consume tobacco to such an extent as to injure their constitutions and induce a state of marasmus often attended with indigestion and diarrhœa; others, especially among the Mussulmans, are addicted to the smoking of narcotic drugs with the hookah, the abuse of which brings on a debilitated state of the system with nervous tremor, and not unfrequently temporary delirium which

¹ Mr. Sibthorpe's Catalogue, Series 8, p. 65, and Series 9, p. 72.

² *Madras*, pp. 95 and 98.

sometimes ends in confirmed mania ; while in all, sooner or later, the habit is followed by emaciation, weakness, indigestion, and fatal diarrhœa.' It is added that the treatment 'is always unsatisfactory, and but seldom followed by a thorough or permanent restoration to health. A course of tonics with gentle alteratives, and careful attention to diet, with the stimulus of wine or spirits, is requisite ; relapses are frequent ; men addicted to narcotics seldom remain longer than a month or two at duty, and are, at last, very generally lost to the service by death or by being discharged.'

In Bengal many well-to-do opium drunkards appear to be fairly nourished, and, escaping serious disease or injuries—under which they would almost certainly succumb—live comfortably into advanced old age. It is said that these individuals generally consume large quantities of milk. This suggests the applicability of milk treatment, strongly recommended by Sir Joseph Fayrer in *diarrhœa alba*, to this kind of intestinal flux.

Ichoræmic Diarrhœa.—It cannot be doubted that certain animal and other poisons, inspired and absorbed by the skin, are eliminated by the bowels. Many students suffer from diarrhœa during the dissecting season. Washing his hands after inspecting the body of a renal case, Mr. Hilton said to me, 'I shall not be able to wash it off ; I shall have to sweat it out.' My own personal experience fully confirms this. I recollect that the first students appointed to dissect at the College of Surgeons were long at work upon a dog. More than one being attacked with severe diarrhœa, the stools had the unmistakably characteristic odour of the animal's skin. My predecessor, as Principal of the Calcutta Medical College, Mr. James Macrae, a remarkably strong and healthy man, having worked very hard in examining the body of a putrid elephant, was attacked with dysentery which proved rapidly fatal. In reports of cases of nearly all diseases, medical and surgical, but especially of the latter, after lithotomy and other capital operations, in disastrous midwifery cases, and where there are compound fractures and large open or sloughy wounds, the words—'the patient was attacked with diarrhœa' [or frequently, in India, 'dysentery'] 'and sank in a few days'—so frequently occur that they might be stereotyped by the printers of medical works. This happens not only in India, where intestinal disease is extremely common, but also by no means rarely in England. I do not think that this point has, up to this time, received the close attention which it deserves by the investigators of septicæmia and hospitalism. It is probable that, in sifting these cases, various causations will be assigned:—thus in class A, say in lithotomy and other bladder cases where intestinal flux often occurs, failure of renal action will have to be considered ; in B, as in cachectic Indians, and other victims of marsh poison and scorbutus, it may be said that death commences at the bowels ; in C we may consider that, towards

the close of exhausting disease, the failure of vitality leaves the patient a ready victim to the most prevailing diseases of the climate or season; in D, as in cases of hepatic abscess, instrumental labour, &c., the flux may be an expression of septicæmia, being an ineffectual effort by the intestinal glands to eliminate the poison, and being evidently associated with other evidences of blood-poisoning.

These have been my readings of many such cases; but, unquestionably, the whole subject demands systematic investigation in all hospitals, at home and abroad.

A full review of the question of the part taken by *Bacteria in Diarrhœa and Dysentery* is given at p. 129 of Fayrer's Lettsomian Lectures. All readers will doubtless view the matter as he and Dr. McConnell do, regarding the presence of bacteria as a natural attendant upon ulceration and sloughing. More lately, Dr. Prior has endeavoured to show, in the 'Centralblatt für Klin. Med.,' No. 17, that a micrococcus is almost certainly the cause of dysentery. He has detected micrococci in enormous numbers not only in the recent stools of a case of dysentery, but also in the juice of the tissues, and in the tissues of the affected intestines themselves. Although such research ought to be encouraged, it carries with it the drawback of reviving views which caused embarrassment to those who first entered upon the quest for the cholera germ. In a very sound notice of this memoir¹ it is observed that 'the alimentary canal is as much outside the organism as is the space which is partly bounded by the skin, and is liable to the action of similar agencies.'

I have noticed *Melæna* (discharge of hæmoglobin from the intestinal canal), under the head of Malarial Cachexia, page 246, see also p. 190.

Intestinal Obstruction.—At a meeting of the Calcutta Medical Society in April 1880, Baboo A. C. Bannerjee exhibited an abrupt *stricture of the jejunum*. The strictured part had the appearance of a healed ulcer. There were marked hypertrophy and dilatation of the muscular coat above the stricture.

A European soldier, at Masulipatam, died from intestinal obstruction. 'For some time previous to death he used to vomit daily large quantities of pure chyle, mixed with a brownish fluid, but without stercoraceous odour. The *Ileum was much contracted, thickened, and semi-cartilaginous to the extent of 3 inches*. The parts near the stricture appeared to have suffered from severe inflammation, as there were numerous adhesions from much lymph thrown out which had become organised. The stricture appeared to be chronic; and, before death, its site could be distinctly felt externally, especially when the bowels were loaded.

Prep. 404 in the C. Medical College Museum Catalogue shows stran-

¹ *M. T.* for 1883, vol. i. p. 508.

gulation of a knuckle of small intestine by an adventitious band occurring in a patient of mine. The strangulated gut is about the size of a hen's egg and of a black colour. Here, as doubtless in many other cases, I was guilty of the blameworthy oversight of not specifying the patient's race.

Morehead also found 'a portion of the small intestine strangulated by old adhesions resulting from former peritonitis.'

In 1868 Dr. Collis reported¹ that he had received at the Calcutta Medical College Museum, a specimen of 'Strangulation of the Ileum caused by twisting on itself.'

A powerful negro in my ward died from displacement of the colon.

Mr. Brockman has reported² an analogous case, *twist of the sigmoid flexure*, occurring in an English gentleman in India, and I heard of another case, the subject of which was also a European officer.

I contributed Prep. 438, in which the mesentery of the sigmoid flexure had undergone contraction and formed that portion of the large intestine into a loop. This was an old, possibly a congenital change. Under some recent cause of bowel irritation, the loop had become distended and the neck twisted. The obstruction having increased, the neck at the entrance point has ulcerated almost across, permitting extravasation into the peritoneal cavity, thus causing fatal general peritonitis.

In the chapter on Dysentery, I have described the case of a native in whom dysenteric thickening of the ascending colon caused obstruction.

A sepy³ had been costive and had complained of abdominal pain and tenderness for a month. He was not much emaciated. He died with symptoms of intestinal obstruction, not having passed a motion for 5 or 6 days, and vomiting incessantly. The peritoneal cavity was almost obliterated by old adhesions. The ascending colon was found to have become reduced to the size of a little finger and atrophied, 'the canal being bounded simply by the longitudinal bands of muscular tissue. The calibre of the gut, in its ascending portion, was reduced to the diameter of a small quill. The small intestine was distended with fluid.' A few years ago, I was deeply concerned in the case of an esteemed retired brother officer who suffered from thickening, doubtless malignant, apparently seated at the highest part of the colic arch. An aggravating circumstance in this case, where obstruction never became extreme, was an irrepressible tendency to use cathartics. The tumour, which felt very like an uncooked sausage, nearly resembled, outwardly, one for which a clever native sub-assistant surgeon

¹ *I. M. G.*, p. 83.

² *Ibid.* for December 1878.

³ I obtained this case from the *I. M. G.*, but failed to note the reporter's name and the volume.

consulted me, greatly fearing that it was malignant. I, however, recommended laxatives, as it could be moulded by the fingers. It was stercoraceous, and disappeared.

Mr. Thomas Maxwell treated a native, *æt.* 60, who had not passed any stool for a week. There was great abdominal distension, and vomiting without stercor had commenced. The case proved to be one of over-distension of the colon by impacted feces, which were removed, under chloroform, by the free injection of warm water through the long tube carried up through the canal to its head.

Prep. 262 in the C. M. C. is '*An Epithelial Cancer of the colon.* Its surface is ragged and uneven. The disease apparently involves the whole of the coats of the gut.'

In England, I saw more than one case fatal by *malignant disease stricturing the junction of the sigmoid flexure and rectum* at O'Beirne's annulus or sphincter, just opposite the brim of the pelvis. I heard, some years ago, of a gentleman who came home from the West Indies with this form of intestinal stricture. In a man, at Guy's, who passed fluid feculent matter with his urine, a huge cloaca had formed by destruction of the left side of the bladder and of the soft structures of the iliac fossa.

Morehead gives a case (at p. 454) of '*obstruction from Colloid cancerous degeneration of the sigmoid flexure.*' The woman had only been a few months in India.

The returns for the 4 years ending 1883 give the following statistics of *Ileus and Obstruction of the Bowels*.—1880, of 11 European soldiers admitted, 6 died; European child, 1 admitted; native soldiers, 13, all fatal; prisoners, 15 admitted, 12 died. 1881.—European soldiers, 3 admitted, 1 died; child, 1 admitted; native soldiers, 11 admitted, 6 died; prisoners, 20 admitted, 15 died. 1882.—European soldiers, 3 admitted, 2 died; child 1, fatal case; native soldiers, 7 admitted, 4 died. 1883.—European soldiers, 1 case fatal; children none; native soldiers, 6 admitted, 4 died; prisoners, 10 admitted, 9 died. No woman was attacked during the 4 years. Neither in India, nor elsewhere, have I seen intussusception or volvulus in a woman.

Intussusception.—Dr. Shirley Deakin performed¹ laparotomy and enterotomy upon a regimental sweeper, *æt.* 20, in whom there was *intussusception of 14 inches of the Ileum* from 19 inches above the ileo-cæcal valve. The patient sank seven hours after the operation.

Ewart has met with *Invagination of the Ileum* consequent upon dysenteric destruction of the ileo-colic valve.

There is a case,² by Mr. T. E. Baker, in which a healthy European infant, 4 months old, died after suffering from intestinal obstruction for three days. About an inch of the ileum and the whole of the colon, to the rectum, were invaginated.

¹ *I. M. G.* for June 1878.

² *I. M. J.*, vol. i., N. S., p. 284.

I treated various forms of intussusception. There were admitted to my native male ward two patients, a Mohammedan and a Hindoo, in both of whom, as in Mr. Baker's case cited above, *a wandering cæcum had become invaginated into the ascending colon*. The preparations are thus described in the Museum Catalogue:—'418. Intussusception of the cæcum into the ascending colon, carrying with it the lower end of the ileum. A portion of the ulcerated and gangrenous gut, invaginated, is exposed. About four inches of the ileum, at the point of invagination, is *in situ*. 419. The exact counterpart of 418, showing the ulcerated and gangrenous invaginated mass completely turned out of the ascending colon which contained it. About four inches of the inferior end of the ileum, at the point of entry, is *in situ*.' For remarks on the tendency of undescended cæca to be included in congenital umbilical herniæ, see the chapter on Hernia, p. 594.

Dr. Herbert Baillie¹ found belladonna answer remarkably well, in cases of intussusception and obstinate constipation, administered in half-grain doses, combined with two or three grains of calomel, in form of pill, given every two or three hours until relief occurs. Three or four pills generally suffice; but he has given as many more, and in no instance has he seen any ill-effects follow.

Dr. Moriarty gives² cases in which the treatment of acute intestinal obstruction, by repeated doses of belladonna, advocated by Dr. Norman Kerr was employed. It may, I venture to think, be questioned whether these cases were not rather extreme examples of constipation, which is a not uncommon condition in the natives of India, than of actual organic obstruction.

Within my experience, scarcely any greater improvement has been made in practice than in the abandonment of feeding by the stomach and of giving cathartics in the treatment of organic intestinal obstruction.

Before I went to India, it was customary to begin with mild aperients and to proceed to drastics. These failing, a pound of quicksilver was 'exhibited,' the patient dying in extreme agony, with an almost bursting abdomen and stercoraceous vomiting. The intestine above the impediment was found inflamed and distended to the very uttermost by a thin feculent cruor and the fluid mercury. In several cases, at Dr. Joseph Ewart's suggestion, I *gave nothing by the mouth*, unless the patient craved for a little milk or water, and nourished by the rectum. In some of my cases, the lesion was absolutely beyond repair; but I am convinced that the principle is absolutely true and valid. One man, in whom there was complete obstruction for many days, suddenly passed an enormous fluid stool, in which gangrenous intestine was looked for in vain, and then speedily recovered.

Early in my Indian career, I attended one of the Peninsular and

¹ *I. M. G.*, vol. v. p. 36.

² *Ibid.* for August 1880.

Oriental Company's medical officers, who, while I knew him—which was for a considerable time after his illness—appeared to be a very healthy gentleman of middle age. He had an acute attack of most intense abdominal pain (it was neither peritonitis nor colic), with constipation and incessant vomiting—not stercoraceous. Except in cases of intussusception, where the symptoms are not generally by any means so violent at the outset, I have never seen any case which so completely realised the old notion of *Passio Iliaca*. Dr. Miller, late of Southsea, recommended a single sedative dose of calomel. When this had been taken, all distressing symptoms rapidly subsided, and he made a good recovery. I think that there must have been temporary obstruction of the small intestine pretty far down.

The returns for 1882 give the following data, under the heading of *Prolapsus Ani*:—Admitted, 15, *European Soldiers*, invalided, 2. Treated: *Soldiers' Children*, 5; *Sepoys*, 8; *Prisoners*, 19.

Ordinary cases of prolapsus ani occur in weakly European and native children, consequent upon chronic bowel complaint, stone, &c. In natives of weak muscular tone, enormous prolapse of the bowel is liable to take place, with or without extensive hæmorrhoidal disease. Driving through a bazar near Howrah one day, I was called into the dwelling of a native writer, in whom there was a recent protrusion of the bowel larger than a foetal head. The mucous membrane distilled a thin fluid very like liquor amnii; around the neck of the tumour were cutaneous excrescences, traces of recovered piles. Against attempts to reduce it *en masse*, the tumour resisted absolutely. I then took his long *cheddar* (several yards of very fine white muslin), and, rolling it into a ball and dipping it in oil, pressed it into the intestinal passage, which formed the most depending portion of the tumour. I then placed him on his elbows and knees, and, pressing the cloth firmly upwards, told him to turn down on his right side. As he did this, the prolapsed bowel suddenly went up, carrying with it my hand and forearm. Then yard after yard of the muslin had to be cautiously brought away. In such extreme cases as this, Mr. John Scott¹ employed the actual cautery with a view to narrow the outlet.

Mr. S. B. Partridge² treated a Mohammedan weaver, *æt.* 40, with an irreducible prolapse of the bowel, the tumour being about the size of the foetal head at full term. The bowel began to come down seven years previously, after a severe attack of dysentery. The tumour was reducible until about two months and ten days previously. From that period, however, all attempts at reduction had proved of no avail. Doubtless these attempts had been of no gentle character. The mass was congested, œdematous, and indurated, and surrounded, about two inches from the central orifice, by an inch-broad ring of

¹ *Bombay Med. and Phys. Trans.*, vol. ii.

² *I. A.*, No. xxvii. p. 237.

ulceration. Under treatment, the œdema gradually subsided, and the ulcerated surface healed, but there still remained so much sub-mucous thickening that reduction was impracticable. Two months after his admission, it was determined to remove the tumour. A slightly-curved needle with an eye near the point, armed with a strong silk ligature, was introduced into the bowel, guided by the left index finger, until its point reached a level above the line of thickening and ulceration, and within about one and a half inches of the position of the external sphincter, the point was then made to perforate the tumour from within outwards, one of the ends of the ligature being retained externally; the needle point was withdrawn and again made to perforate the tumour, on the same level, at a short distance from the previous perforation, in a line forming a portion of a circumference of the intestinal canal, the other end of the ligature was then unthreaded and retained externally. The needle remaining *in situ*, a fresh ligature was threaded, one end retained externally, the needle point withdrawn, and a third perforation made in the same continuous circumferential line. This process was repeated until the whole circumference of the bowel had been apportioned into segments, each embraced within its loop of ligature with the ends of the loop external. These ends were next firmly tied, and the tumour effectually strangulated, without, however, occluding the anal aperture. The tumour was then removed by scalpel, about an inch below the line of strangulation. Some bleeding was arrested by fifteen ligatures. A narrow *cul-de-sac* was found in front of the central canal of the tumour. This was evidently the lower end of the recto-vesical pouch of peritoneum. For fourteen days, he appeared to do wonderfully well. There was no abdominal pain or tenderness. The bladder acted naturally, the separation of the strangulated portion of the bowel proceeded most satisfactorily; and, ultimately, the bowels were moved freely and without difficulty. Now, however, tetanus set in and proved fatal in thirteen days. Previous to death, the slough had entirely separated, leaving a clean, healthy, granulating surface. The pelvic peritoneum was found to be perfectly healthy; the cut extremity of the recto-vesical pouch had completely closed. In itself, therefore, this operation was successful.

Congenital Imperforation of the Rectum and Anus.

Indian medical literature embodies a very considerable number of cases of the various forms of this class of congenital defect. Assistant-Surgeon G. C. Dey, of Raipur, tells us that a child born without an anus is designated by the natives a 'Paigumber, or messenger from God.' In 1854, I published¹ a report upon this subject, founded upon two cases of *Atresia Recti Urethralis*, upon which I operated at Chit-

¹ *I. A.*, No. 1, p. 297.

tagong. Four years later, Mr. W. A. Green, then of Dacca, published¹ an account of eight cases which had come under his own and his predecessor's care at that place. Mr. Green remarks that, as Dr. Johrer, of Vienna, met with two cases out of 50,000 new-born children, and as Dr. Collins, of Dublin, observed one case out of 16,634 children, 'the individual experience of Dr. Chevers and myself, at Chittagong and Dacca, has been comparatively large.' At Chittagong, where the applications for surgical relief were by no means numerous, *five* cases came under treatment in fifteen years. My report contains a rather full analysis of the cases of congenital closure of the bowel, *in males*, which were then on record. Having, at that time, the case of a native girl under treatment, it was my purpose to classify these defects as they occur *in females*; but I failed to do this. The following malformations have been observed in males:—

I. Obliteration of the duodenum; jejunum; ileum; ileo-colic-valve; colon, either at its ascending portion or sigmoid flexure, or of the upper portion of the rectum, reducing the intestinal canal below to a mere cord. As a general rule, the lower part of the rectum and anus are developed in these cases, but are liable to be found strictured or much contracted.

II. Obliteration of the rectum, the colon communicating with the bladder or membranous portion of the urethra.

I wrote long ago that 'it appears that, in the whole of this class of cases, the normal pelvic portion of the intestinal canal, and the anus, are entirely absent.' I see, however, that, recently, Dr. William Craig has described and figured² the case of a child, which lived eighteen days, passing *fæces* regularly by the penis with intense agony, in which there was a rectum opening into the membranous portion of the urethra by a passage about as narrow as the urethra itself. The second of my cases (see p. 586) greatly resembled this one, save that, in mine, there was no rectum or anus, while in Dr. Craig's patient there was an anal *cul de sac*, of sufficient depth to admit the greater part of the distal phalanx of the little finger.

There is a class of cases in which the rectum and anus are not developed, and in which no communication of the intestine with the urinary organs is observed. Whether these represent a distinct variety, or whether a very narrow communication with the bladder or urethra may not have existed unnoticed in all, appears uncertain.

There are instances in which the rectum terminates in front of the scrotum, or under the glans penis, in a canal formed in the substance of the scrotal raphe.

III. Obliteration or extreme contraction of the anal orifice, independently of other defects in the intestinal canal. It will be found that

¹ *I. A.*, No. 9, p. 269.

² *Edinburgh Medical Journal*, March 1884, p. 800.

I have given references to cases of each of the above defects. In Dr. Green's eight cases and in three preparations in the Calcutta Medical College Museum, the following malformations occurred.

Male.

1. No anal orifice. Perineal incision ineffectual. Colon opened in the left iliac fossa. Recovery. (*Green.*)

2. Fæces passing by urethra. No anus. Perineal incision unsuccessful. (*Green.*)

3. A small elongated sinuous opening, just behind the scrotum, leading to the rectum. No natural anus. Perineal incision successful until the child was taken away in six days. (*Green.*)

4. A very contracted orifice (congenital) in place of anus. Opening enlarged. Did well for fifteen days and was taken away. (*Green.*)

5. The urethra opened behind the glans penis, and meconium was passed by this channel. Perineal operation unsuccessful. (*Museum, Prep. 446.*)

6. Rectum developed to within an inch of the usual point of exit. Bowel enormously dilated for four inches above the blind end. (*Museum, Prep. 447.*)

7. Arrest of development of the rectum has taken place within a quarter of an inch above the usual site of the anus. Intestine dilated. (*Museum, Prep. 448.*)

Female.

8. No anal opening. Fæces passed apparently by a sinus communicating with the vagina. A director was inserted into the sinus through the vagina, and being made to project towards the proper site of the anus, was cut down upon and an artificial anus formed. Fæces continued to pass for two years by both openings. Then, fæces passing by the vagina, the artificial opening was re-made; but the parents neglected to keep it open. The child died in its fourth year, fæces passing by the vagina. (*Wise and Green.*)

9. No anus, a slight depression in its place. Perineal incision. Meconium evacuated by canula. Death in six days. (*Green.*)

10. A female child aged six months. Fæces passed per vaginam through a passage beneath its tract opening at the fossa navicularis. Natural site of the anus bulged in the acts of crying and defecation. Operation as in Case 8. The *large ascites canula* was used in keeping the artificial passage open. Hence, I consider, the promising result. Child taken away, but continued well on the sixteenth day after the operation.

11. Native girl of five. Malformation and operation as in Case 10. *Large canula* employed. The child was removed as usual; but it was reported that, five weeks afterwards, she was quite well, the evacuations being made through the newly-made anus.

Male.

It was rumoured that a perfectly successful operation had been performed by a French surgeon at Chandernagore.

The following is an abstract of my own cases. Native boy five days old. No trace of anus. Meconium in small quantities passed frequently by urethra with urine and flatus. Incision. *Cul de sac* of colon opened by hydrocele trocar. Escape of meconium. The child died on the third day. It was brought late; and, although the artificial opening was free, it was not large enough.

A native infant, also five days old. Condition of external pelvic organs nearly as in last case. Incision and introduction of small trocar. A very copious discharge of meconium gushed through the canula, with evident relief to the child. On the third day, the artificial opening had to be enlarged. Latterly, the stools were free, usually somewhat diarrhœal. The child gradually emaciated and declined, and died thirteen days after the operation. *P.M.* General peritonitis. No trace of inflammation or purulent or feculent infiltration in the course of the incision. The very capacious colon terminated in a *cul de sac* a little below the level of the sacral promontory. A free opening had been made by the trocar into this pouch, exactly at its lowest part. A curved probe, passed into the urethra, entered the bladder or the bowel with equal freedom—a narrow canal passing from the fore part of the intestinal *cul de sac* into the neck of the bladder or membranous portion of the urethra.¹ I doubt whether my surgical observations upon this interesting class of cases, made upwards of thirty years ago, would now deserve attention; but there is much in Green's paper and my own, not cited here, which, I submit, ought to be seen by one who makes a special study of this subject. I concluded my remarks with an observation which has ever since retained a place in my mind. If again called upon to perform the operation, I should, after making a small incision in the integument, and exploring, introduce a trocar, about two inches long, and nearly half an inch in circumference, with great caution upwards towards the promontory of the sacrum. Once in the intestine, the canula should be retained for at least a week, and then, as Copland Hutchinson recommended, a portion of gum-elastic catheter should be substituted, and, when the tube can be dispensed with, a sponge tent or a piece of bougie should be worn twelve out of the twenty-four hours. The pressure of a canula of *sufficient calibre*, and properly adapted for remaining in the intestine [its upper edge should be rounded], must

¹ Unfortunately, the examination being interrupted, the specimen was placed in bad spirit, so that I failed to notice the precise spot at which this intestinal passage entered the urinary canal. It, however, appeared to open just anterior to the prostate.

evidently afford the best protection attainable against the occurrence of feculent effusion and infiltration in and behind the peritoneum. Subsequently, when the divided tissues, throughout the entire tract, had become firmly agglutinated, any moderate degree of dilatation might be undertaken with safety.

The following cases, illustrative of congenitally imperforate anus, as occurring in India, will well repay the attention of those who may wish to study this condition :—

A native child, one day old. No appearance of an anus. An opening was made to the depth of about three-quarters of an inch, when meconium poured out in a full stream. Child did well. (James MacRae, 'I. M. J.,' vol. v., N. S., p. 487.)

Child operated upon, on the fourth day, for imperforate anus, by opening the ileum near the cæcum in the groin. The child survived the operation six days. ('I. A.,' No. 21, p. 296.)

Child born in the Calcutta Medical College Hospital with imperforate anus. The penis was small; the scrotum was represented by two folds of skin, resembling labia, without testes, separated by a fissure a quarter of an inch wide, at the bottom of which the ordinary skin of the perineum is visible. Sigmoid flexure, enormously distended and ending in a constricted portion, about one-third of an inch long and one-sixth of an inch wide, continuous with the superior and inferior part of the bladder. The child had never made water. The bladder contained about half an ounce of urine which, under pressure, escaped readily through the urethra. Air cannot be forced from the bladder into the sigmoid, nor meconium from the latter into the former. A large convoluted ureter leads from the posterior part of the bladder on the left side to the left kidney. From the lower angle of the latter, a band of glandular substance passes in front of the aorta and terminates in a mass, not much larger than a pea, partly glandular, partly vesicular, which is the only representative of the right kidney. No testes, ovaries, or uterus existed. (Dr. Charles, *ibid.* p. 303.)

A child, at Delhi, twenty hours old, had no anus, but, in its place, there was a slightly elevated piece of skin continuous with the raphe. An opening to the depth of an inch not reaching the bowel, Amussat's operation for colotomy was performed. The child did well for five or six days, was removed by his parents, and died convulsed about a fortnight later. (Ashootosh Goopto, 'I. M. G.,' vol. ii. p. 246.)

A male child, 7 days old, was brought to the Azimgunge Dispensary with imperforate anus and hypospadias, with contraction of the orifice of the urethra. By dissection to the depth of an inch, the bowel was reached and opened. Death from peritonitis, enteritis, and commencing pelvic cellulitis in 24 hours. (S. C. Chatterjee, B.A., M.B., 'I. M. G.,' vol. vi. p. 54.)

Assistant-Surgeon M. S. Huq has recorded three cases, all in male

infants.¹ (1) There was a sense of distension in the perineum, but no anus. At the depth of two inches, the bowel was found and opened. Cure in eight days. (2) A well-formed anus which ended, nearly three inches up, in a closed sac. A knife passed up gave vent to a large quantity of meconium. Discharged well on the sixteenth day. (3) A circular violet-coloured spot at the situation of the anus. An incision gave free discharge to meconium. Recovery in six days. One patient subsequently died of some other disease; the others continued in every respect healthy.

Baboo G. C. Dey describes² a child the subject of complicated deformity of the lower part of the body. The large intestines, omentum, and bladder, were entirely wanting. The small intestines terminated, below the umbilicus, by a tubular projection, from which yellow feculent matter escaped.

Hæmorrhoids occur in India nearly as they do at home, save that, in consequence of the greater prevalence of bowel-complaint there, they are probably more common in the East.

The returns show that, in 1882, there were 7 *European Soldiers* invalided, out of 461 treated, for piles. The other admissions were: *European Women*, 14; *Sepoys*, 206; *Prisoners*, 381. Occasionally the masses become very large and heavy, and are attended with very troublesome prolapse of the bowel, especially in those who have suffered much from dysentery and diarrhœa. In contemplating an operation for their removal, we should consider that a pile (apart from prolapse of the gut) is not a mere disease and injury to the system as an ingrowing toe-nail is—it often has its function. A very old Indian resident, still living, a man of enormous bulk, said to me, ‘My piles have kept me alive.’ This is not necessarily the case, as other old Indian contemporaries of his remained equally bulky and healthy without piles. Still, there is truth in his very popular idea. Piles, which cease to be required as a means of giving vent to surplus portal blood or hæmoglobin, generally waste, and completely, or in a great measure, cure themselves; or, where the effete mass is bulky, they may be safely removed. An eminent Calcutta medical man complained to me greatly of the ill effect which, he considered, the ablation of a large hæmorrhoidal mass had on his portal system. But he lived subsequently, in India and at home, for many years, holding an important appointment for a long time after his return to this country.

Mr. C. Macnamara has related³ the case of a young man in whom the ligature of bleeding piles was followed, on the third day, by tetanus, which was fatal in 48 hours. *Vide antea*, pp. 419 and 583.

In 1857, an elderly and corpulent officer, whom I knew at Chittagong, came a long voyage by boat to Calcutta, and sent for me. As I

¹ , *M. G.* for June, 1872. ² *Ibid.* for 1874, p. 10. ³ *Ibid.* vol. viii. p. 295.

did not practise at that time, the late Dr. Harrison accompanied me. There was incontinence of urine and an enormously distended bladder, projecting the abdomen as in pregnancy. When a prostate catheter was passed, we saw the prominent belly subside. The obstruction was caused by the pressure of a huge hæmorrhoidal mass. His bladder never recovered its contractile power; and, although he received close attention, the urine became ammoniacal, loaded with mucus, and fetid. Death occurred in about twenty days.

There is a class of native practitioners with whom the treatment of hæmorrhoids is a speciality. There is an obscure mention of the practice, at Dacca, of removing piles by the application of 'corrosive substances' [arsenic ?]¹ Dr. Percival Lord gave² a full description of the mode adopted at Dera Ismael Khan. A needle, holding a ligature, was passed through the hæmorrhoid, which was then tied tightly round the neck. The tumour may then be sprinkled over with *sunkia* (white oxide of arsenic). It was stated that the mass generally fell within seven days.

Dr. Herbert Baillie employed undiluted carbolic acid in a few cases of internal piles with most satisfactory results, the patients having been speedily relieved, certainly not with more pain than when the nitric acid has been used, and with no abrasion of the mucous membrane, which, after a time, appeared braced and healthy.³

Fistula in Ano is not common in Lower Bengal. The returns for all India in 1882 give the following data: *European Soldiers* treated, 67, invalided, 3. Admissions: *Soldiers' Wives*, none; *Sepoys*, 56; *Prisoners*, 50 with 2 deaths. When occurring in Europeans, I have more than once heard ischio-rectal abscess attributed to sitting on damp gratings on the decks of passenger ships.

Dr. W. J. Moore has noticed⁴ that fistula, hæmorrhoids, and rectal diseases generally appear to be more than usually common in various parts of Rajpootana—notably in Marwar. This appears to result from the general practice of camel-riding. A camel saddle, with its numerous padded coverings, maintains the parts concerned moist and heated, while the continuous jerking up and down, consequent on the peculiar gait of the camel, of course adds to the congestion of the vessels and structures surrounding the rectum. Assist. Surg. Bhowani Das has made the curious but debatable observation⁵ that *oxyuris vermicularis* is to be accounted as one of the causes of fistula in ano.

Treatment.—Sir J. Fayrer had cases of debilitated patients, who could not spare blood, in which the écraseur was employed with advantage.⁶ Dr. Herbert Baillie has insisted⁷ that division of the sphincter

¹ *I. M. J.*, vol. ii. p. 140.

² *Ibid.* N. S., vol. iii. p. 400.

³ *I. M. G.*, vol. v. p. 210.

⁴ *I. A.*, No. 16, p. 300.

⁵ *I. M. G.*, vol. iv. p. 259.

⁶ *I. M. G.*, July 1876.

⁷ *I. M. G.*, vol. iv. p. 184.

ani is absolutely necessary in *every* case of rectal abscess, to avoid the contingency of subsequent fistula. The muscle should be divided when the abscess is opened; and, as the left fore-finger, well-oiled, should always explore the abscess and search for sinuses, the sphincter can then be put upon the stretch and easily divided. He has seen weeks lost and much unnecessary suffering caused by want of attention to this particular. Still, there are cases in which, when it is done early enough, opening the abscess suffices.

Anal Fissure.—An ulcer, situated where mucous membrane and skin meet, which is made, by contraction of the sphincter, to appear as a cleft, is rather frequent in India. It may not unfrequently be healed by the use of rhatany or argenti nitras, early employed, but it is sometimes needful to incise.

Mr. William White has left a useful paper on this condition.¹ He deprecated the division of the sphincter, and recommended lighter incisions, not extending deeper than the diseased mucous membrane, which he had employed with immediate and permanent relief in two European soldiers.

I did not see *Malignant Stricture occurring low down in the Rectum*; but epithelial cancer occurs (Ewart's Catalogue, Prep. 255). Dr. Collis described as follows a specimen of stricture of the rectum, subsequently received at the museum²:—‘Uterus, part of the bladder, vagina, and rectum of a patient who was admitted with stricture of the rectum. A large broken-down cavity exists between the vagina and rectum, communicating with the latter through a ragged opening in the anterior wall of the gut, and above with the recto-uterine pouch of peritoneum. Scrapings, both from the mucous membrane and the cut edge of the strictured gut, contained little besides fat globules, granules, and twisted fibres like those of yellow elastic tissue. No nucleated cells were found.’

Stricture of the Lower Bowel, caused by Dysenteric Ulceration, is liable to occur. A very distinguished administrative medical officer was long haunted and rendered miserable by the idea that he had malignant stricture—he said that the disease was in his family. I considered that it was due to dysenteric lesion. I understood that it yielded to ordinary treatment at home.

The lower part of the bowel was greatly contracted in a young European in my ward who lost immense portions of the interior of the intestine by dysenteric sloughing. He did tolerably well, by the constant use of rectal bougies, until I lost sight of him.

Dr. Ewart received, at the Calcutta M. C. Museum, ‘a very large *Rectal Polypus* from a native named Buckshoo, *æt.* 63. The pedicle was deligated by Mr. S. B. Partridge. The tumour fell off on the fifth day. The patient made a speedy convalescence.

¹ *I. A.*, No. 5, p. 117.

² Ewart, *I. A.*, No. 21, p. 304.

Prep. 64, Series 8, in the Madras Catalogue, is 'portion of the rectum with a polypus attached to its lining membrane. From a (doubtless native) subject in the M. C. dissecting-room.

Colic.—I notice, in the old regimental returns, that colic, attributed to drinking palm toddy, used to be very prevalent among the European soldiers. In 1883, the statistics of colic were : European soldiers, 417 cases ; native soldiers, 842 cases, 4 deaths ; prisoners, 803 cases, 2 fatal ; European women, 35 ; children 10, with one death.

Dr. J. C. G. Carmichael has made¹ some useful practical remarks upon malingerers in native regiments. He says that everyone must be acquainted with the innumerable cases of pain in the abdomen which come to hospital every day of the week except Thursdays and Sundays ; nine-tenths of these are malingerers. ' Few or none come on the days when there are no parades.' Contrasting the symptoms of colic and other abdominal disorders with those complained of by malingerers, he shows that the bowels may be regular or not, pressure is said to increase pain, the tongue is clean, the countenance clear. The men come to hospital at parade-time or when on duty ; they exaggerate greatly and say they have almost any pain you like to mention. The temperature and pulse are normal, and there is no vomiting.

Colic is rather prevalent among rich natives in Lower Bengal. I was, for a considerable time, much distressed by a neurosis of colic. At first, it occurred towards bed-time as a dull sensation of weight and distension in the sigmoid flexure. This was hardly pain, but it caused much distress. On a morning when I was about to leave for England, I had driven down to my hospital, had breakfasted, and was about to make some calls, when I was told that my coachman (a Mohammedan whose mode of life was as different as possible from my own) was ill. My first impulse was one of anger, but I found the man in the hall, suffering intensely from colic. I immediately went upstairs and mixed some medicine for him. Before I could send it down, I was attacked with most agonising colic. In my mind, this singular incidence of the neurosis and alteration in its character is not easily accounted for. My work, at that time, was always somewhat beyond my physical power. In making over charge, there were anxiety and additional work. Still, these causes of lowered innervation are scarcely enough to account for the sudden paroxysm. When I first went to the man, I thought that he was malingering ; but, when I saw the sweat rolling down his face, and observed the other signs of unmistakable physical agony, I at once recognised the reality of his suffering. Still I viewed him unconcernedly as an ordinary patient. Why I began to suffer almost as soon as I perceived his suffering is a problem of which my pathology fails to give a demon-

¹ *I. M. G.*, vol. viii. p. 148.

strative solution. I am confident that this was no mere coincidence. I had merely looked at the man, and hastened away when the pain struck me as if I had been shot through. Had this happened to an hysterical woman, I could better have understood it; but that the nervous system of a medical man, in charge of a large hospital, should be thus sympathetic is curious. The remainder of the history may possibly afford an explanation. While I was in England, the neurosis came back repeatedly, resuming its original moderate character; but it was distinctly aggravated on returning to India. I then suffered very severely several times, but never to extreme agony. On the last occasion, I was in great pain, but not such as to explain the profuse sweating and singular distress of mind. I think it not improbable that I and my servant were the subjects of a phase of masked paludal fever congeneric to the hill colic of Webb, described at pages 189 and 276. The attacks were not periodic. They spontaneously ceased to recur after a course of a year and a half. In another case, I would give quinine freely in conjunction with ordinary means.

In November, 1834, Mr. Raleigh treated, in Calcutta, several cases of colic which he ascribed to 'checked hepatic secretion;' but, as almost all the patients came from one ship, he allowed that it was natural to conclude that 'there may have been something impure about the vessels in which their food or drink were kept.' These cases were not invariably attended with constipation.¹ As these cases occurred in sailors, their perusal calls to mind M. Boudin's description of '*Colique végétale*'² as being very prevalent in French ships on the coasts of Scinde, the Persian Gulf, the Ganges, and Bay of Bengal. But, in Mr. Raleigh's patients, there was nothing of the serious after-symptoms, 'emaciation, tremors, paralysis, delirium, and convulsion,' observed by the French medical men. Morehead never saw colique végétale in Bombay, nor did I in Calcutta. *Vide Lead Poisoning and Colic in India*, p. 293 of my 'Medical Jurisprudence for India.'

In 1834, Mr. D. Stewart reported³ that several cases of Colica Pictorum had come to the Seamen's Hospital, Howrah, from a vessel lately from England. These men had lived for about four months upon salt meat which was transferred once a fortnight from the barrels in which it was stored into a large leaden tank, from which the daily rations were served out. The tank was said to have been carefully cleaned out on these occasions; but a crust of white lead was always found to have accumulated in the intervals. Twenty cases of violent colic came on nearly at the same time, nine of which were fatal.

In April, 1836, there was read, at the Calcutta Medical and Physical Society, a paper, which Mr. Twining had not completed at the time of his death, on some of the consequences arising from the use of lead for

¹ *I. M. J.*, vol. ii. p. 52.

² Morehead, p. 455.

³ *C. M. T.*, vol. vii., part 2, p. 442.

various purposes on board ship. Twenty-two sailors from the ship mentioned in Mr. Stewart's report, cited above, came under Mr. Twining's care. In another ship, 18 of the crew suffered from painter's colic on the voyage out. Sir W. O. Shaughnessy said that arrack frequently contained lead and often traces of copper. It appeared clear that the disease arose from the use of leaden receptacles, for the water and meat, which had been long previously prohibited in the navy.¹

Hernia.

The returns for the four years ending 1883 give the following statistics of hernia :—

1880.				
European Soldiers.	.	.	.	85 admitted, 1 death.
„ Women.	.	.	.	0 admitted.
„ Children	.	.	.	2 admitted, 1 death.
Native Soldiers	.	.	.	60 admitted, 2 deaths.
Prisoners	.	.	.	54 admitted, 2 deaths.

1881.				
European Soldiers.	.	.	.	103 admitted, 1 death.
„ Women.	.	.	.	2 admitted.
„ Children	.	.	.	1 admitted.
Native Soldiers	.	.	.	36 admitted, 1 death.
Prisoners	.	.	.	43 admitted.

1882.				
European Soldiers.	.	.	.	95 admitted.
„ Women.	.	.	.	1 admitted.
„ Children	.	.	.	6 admitted.
Native Soldiers	.	.	.	52 admitted.
Prisoners	.	.	.	58 admitted, 5 deaths.

1883.				
European Soldiers.	.	.	.	76 admitted.
„ Women.	.	.	.	3 admitted.
„ Children	.	.	.	2 admitted.
Native Soldiers	.	.	.	24 admitted.
Prisoners	.	.	.	39 admitted.

Traumatic Hernia of the Stomach through the Diaphragm occurred in the cases of Chief Justice Norman, whose diaphragm was wounded by the downward stroke of a long Afghan knife which entered near the posterior axillary fold; and of a workman who fell from the top of a high building.

Preparation 463, in Ewart's Catalogue, is described as 'A diaphragmatic hernia. The protruded intestine, about the size of a man's

¹ *I. M. J.*, N. S., vol. i. pp. 278-91.

fist, is *in situ*. On the thoracic side, it is covered with peritoneum and pleura. The altered and thickened pleura is opened, showing that, beyond a little opacity, there is scarcely any change in the peritoneal investment. The patient was a prisoner in the Chupra Jail. He had been very severely beaten with fists and *lattees* (heavy bamboos) when caught in the act of stealing. In consequence of this, he remained long in hospital, complaining of pains about the epigastrium, body and limbs; but there were no characteristic indications of hernia. Recovering from these, he was discharged, but returned soon afterwards, suffering from dysentery, from which he died.'

Umbilical and Ventral Herniæ are rather common in India, especially in multiparæ. I used to see a cat which appeared to carry her abdominal viscera on a lower deck nearly as capacious as her body.

In the absence of the colleague to whom the case would otherwise have fallen, I attended a newly born native female infant with what had somewhat the appearance of an umbilical hernia. There was, at the usual situation of the navel, a globular protrusion nearly as large as the child's head. Its covering was a pellucid membrane, very like the tissue of an india-rubber balloon. The whole protrusion was of a deep mulberry colour. As there was evidently strangulation, threatening gangrene, I relieved the stricture, reduced the bowel, and brought the integument together by interrupted suture. In a little while, the bowels were moved slightly. The child died in about thirty-six hours. I found that, judging by its great congestion, the cæcum, which had probably never reached its normal position, together with a large portion of small intestine, had formed the contents of the sac.

A similar case of Mr. Godlee's has been reported.¹ A male child 48 hours old, had a globular or rather ovoid tumour at the umbilicus 'of the size of a large Tangerine orange.' It was covered by a raw surface partly formed of imperfect granulations, and partly of horny flakes of dried umbilical tissue. The child continued to be sick, but the bowels acted from time to time. Fifteen days after admission, the bowels being confined and vomiting continuing, reduction was effected by incision. The bowel was adherent to the sac. The opening was closed with stout silver wire and fine cat-gut sutures. The child died within 48 hours. This case singularly resembled my own, inasmuch as the intestine contained in the tumour proved to be the cæcum and vermiform appendix together with the end of the ileum. On examination, p.-m., it was found that the cæcum, about four and a half inches of large intestine, and about two inches of ileum had formed the contents of the sac. 'The meso-cæcum was four inches and a half long, and indeed, the cæcum and the ascending colon seemed to be fixed to the right border of the mesentery, and were in no way attached to the iliac fossa. The fact that unattached cæca were found in the umbilical

¹ *M. T.*, vol. i. for 1883, p. 640.

sac both in Mr. Godlee's case and in my own appears to indicate the existence of a hitherto unobserved law, in these cases, under which the cæcum &c. are prevented from descending, as foetal development advances, from their original umbilical position.

At a meeting of the Calcutta Medical Society in November 1880, Dr. B. L. Chuckerbutty described the case of a new-born infant which had about four feet of small intestine, with its mesentery, protruding through an aperture below the navel. The umbilical cord was just above it, separated from the lower opening by a thin membranous partition. The pedicle of the protrusion adhered to almost all sides of the aperture except at its lower part, through which a small portion of the gut issued and receded with the expiration and inspiration respectively. The intestine was full of meconium and congested.' [It is evident that the membranous sac which had enveloped the protruding intestine had ruptured.—N. C.] An ineffectual attempt was made to reduce the gut by operation, and the child died in three hours. Dr. Chuckerbutty considered that 'the gut was developed outside the abdominal wall from the very beginning of the developmental process. Dr. Harvey remarked that the intestinal tube lies outside the abdominal cavity till the end of the third month of intra-uterine life. This appeared to be a case of both defective development of the abdominal wall, and imperfect retraction of the intestine. It might be called *ectopia-intestinalis*.'

Dr. Rennie has published, in the 28th number of the China Customs Medical Reports, the case of a native infant twenty-seven days old. Its mother having died, it had been improperly fed. It was troubled with flatulence, and cried almost incessantly. An umbilical hernia, about the size of an orange, was noticed. After a few hours, this burst, and the greater part of the small intestines passed out. They were found lying on the abdomen deeply congested, distended with gas, and smeared with a loose motion recently passed. Having been cleansed with a weak carbolic solution and relieved of some of the flatus by a small aspirator needle, they were returned through the opening, which it was found necessary to enlarge, and the wound stitched up with carbolised silk. The child died in eight hours.

Baboo S. C. Chatterjee¹ attended a native lady whose premature (seven months and two weeks) child had 'a goodly portion of the intestines' [?] 'protruding at the navel.'

Dr. Evans described² the case of a native female child of Calcutta, about five and a half years of age, otherwise apparently well formed, in whom there was an abdominal sac about six inches long and four broad, which, when distended, was of a lengthened ovate form. It occupied the median line, commencing at a hollow in the lower part of the chest, about two inches below the sterno-clavicular articulations

¹ *I. M. G.* for February 1879,

² *I. M. J.*, vol. i. p. 319.

and descending till it came on a line with the pubes. The integument was much attenuated, assuming the appearance of the bag of the adjutant bird. Upon it was seen the cicatrix of the umbilicus. The contents were reducible into the abdominal cavity. In the recumbent position also the stomach returned into the abdomen, leaving the small intestines clearly distinguishable to the touch, and their convolutions were visible through the thin integuments. It appeared, from the high position of the tumour, that the stomach was forced through the diaphragm and into the chest before it passed through the external opening. This idea appeared to be strengthened by the statement that the original defect and protrusion were but small and high up at the time of the child's birth, the increase having since gradually taken place downwards. The tumour, at other times flaccid and comparatively small, became much distended after taking food. The sternum was 'defective,' but its precise condition is not described. It left a triangular chasm immediately above, but unconnected with the tumour. In this hollow, there was a strong vibratory pulsatory movement. To the sight, this was painfully disagreeable, as the apparent labourings of the vessel, which was very superficial, seemed momentarily to threaten rupture. This pulsation and that at the wrist were synchronous—104. An admirable sketch illustrates the report.

Prep. 168, S. 8, in Mr. Sibthorpe's Madras Catalogue, is from a native man, in whom a barber cut into an umbilical hernia which had become inflamed. He was admitted with a fecal fistula. The bowel having become bound down to the edges of the opening, all the feces were discharged through it for eight and a half months, when the bowel became prolapsed and strangulated. After ineffectual attempts to reduce the gut, which was very friable, an incision was made into the rigid edge of the opening, and reduction was effected with some difficulty. Death from peritonitis in three days. The bowel which had protruded, and which was atrophied, was a portion of ileum about two and a half feet above the ileo-cæcal valve.

On recalling to mind a rather considerable number of cases of umbilical hernia, both in infants and in adults, it appears to me probable that *all cases of spontaneous umbilical hernia arise from varying degrees of congenital defect.*

Radical Cure of Ventral Hernia.—In a native woman who was outcasted on account of her disease, there was a hernia as large as a full-grown foetal head, protruding two inches above the umbilicus. A small protrusion had been noticed a year previously. The hernia was easily reducible through an oval ring three inches by two wide. Dr. R. F. Hutchinson determined upon attempting Fayrer's operation. He prepared a flat plug two inches broad and three-quarters of an inch thick, with apertures for two ligatures. Passing the bag of the hernia through the ring downwards and outwards, so as to avoid the umbi-

licus, he fixed it with two ligatures, the needle being brought out in four different spots; the plug was then introduced and tied in, and a broad bandage applied over it. On the fifth day, as the ligatures had fairly caused suppuration, the plug was removed. A pucker was presented in the abdominal integuments, the size of an adult's mouth, and below it there was a great deal of thickening. A pad was put on and the patient was kept strictly on her back. There was not any untoward symptom. The walls of the invagination, without being made raw or scarified, adhered firmly, and the pucker rapidly contracted, and, in twenty-three days after the operation, presented the appearance of a small umbilicus. The ring had contracted into an oval of one inch by half an inch, so that she was virtually cured, and, with the protection of a leather belt, could again enter the society from which she had been debarred.¹

Mr. Thomas Wilkinson King wrote a paper to show that much of the *Strangulated Hernia* which occurs in London is in worn-out and intemperate people, in whom hepatic and other visceral disease leads to portal obstruction and, consequently, congestion of the prolapsed bowel. He also showed that a very large proportion of cases of strangulation and incarceration occur in cold weather, when the tendency to portal congestion is greatest. I am sure that there is valid truth in this observation, and I think that it goes far to explain the fact that, although there is a great deal of hernia in the hot climate of Lower Bengal, strangulation is not a frequent accident there among Europeans.

Writing forty-nine years ago,² the late Dr. Henry Goodeve remarked that, 'Strangulated hernia is a rare complaint amongst the natives of India.' Later experience has shown that it rather frequently occurs in ill-fed natives; but is not, I think, so common as among the poor of London.

The following notes are sufficiently illustrative of the frequency with which strangulated hernia occurs in the great hospitals of India. In 1875, Dr. Cayley operated, at the Calcutta Native Hospital, on 7 natives, with 4 deaths; and used the cold taxis with success in 7 other natives. In five years, Dr. Kenneth MacLeod operated in 18 cases with 9 deaths.³ In 1883, 23 natives and 7 Europeans were admitted to the General Hospital, Madras, with hernia; of these 12 were strangulated or obstructed, 6 of whom died.

Sir Joseph Fayrer has given very full reports of cases of strangulated hernia occurring in Bengal.⁴ The following excellent practical remarks appeared in one of his early reports.⁵ 'These cases illustrate

¹ *I. M. G.*, vol. iii. p. 32.

² *C. M. T.*, vol. viii. part 2, p. ccc.

³ *Operative Surgery in the Calcutta Medical College Hospital.*

⁴ *Clinical Surgery in India*, p. 432, and *Clinical and Pathological Observations in India*, p. 333.

⁵ *I. A.*, No. xvi. p. 322.

the advantage of operating early in strangulated hernia. I am convinced that, however great the danger of delay may be in the European, it is greater in the native of India. I am satisfied that early operation is most conducive to safety. A native is brought to the hospital with strangulated inguinal hernia: you are told that it has been irreducible for some hours; that, for two days or more, he has had pain, sickness, and constipation; you try to reduce it under chloroform and fail, but you are perhaps led, from the absence of apparent urgency, to delay the operation; you find that there is abdominal pain, but that it is not so intense as you would expect; there is nausea, but not decided vomiting; the bowels are constipated, but a small quantity of feculent matter has been recently passed. The skin perhaps is cool, the pulse feeble—the native pulse is often feeble in health—the tongue is clean, and the countenance is expressive of nothing more than a native can assume for the most trivial ailment. You think that you are not justified in opening a man's abdominal cavity on such evidence as this, you order ice to the tumour, perhaps an enema, and wait. The next time you see him, probably being sent for before your next ordinary visit, you find a change, his pulse has become quicker, his voice more hollow and feeble, his skin cold, he is in a general state of depression, and you then either recognise that he is sinking, or that he is so feeble that he is hardly able to bear the operation, which perhaps comes too late to save him.'

'The natives of India, especially the wretched poor in Calcutta, are extremely prone to pass into this state without any of those demonstrations of an asthenic character that, in the European, or even the vigorous inhabitant of the North-West, would indicate strangulation, inflammation, and approaching gangrene. The condition of strangulation is prone to pass rapidly into that of gangrene, and it is most necessary to recognise this and operate early. I have remarked also the liability to suffer from inflammation and suppuration in the scrotum after the operation, owing no doubt to irritation of the unreduced sac; this, to some extent, is increased by the manipulation the scrotum and sac undergo in attempts to reduce the hernia. In a large proportion of the natives of India, the scrotum, the testicle, and the irreducible sac of a hernia, where there is this complication, are very prone to hypertrophy and thickening, and, in this condition, no doubt, they are more likely to take on suppurative inflammation when irritated, than if in the normal state. Suppuration in the sac after operation for strangulated hernia, I believe to be a frequent cause of death, and it should therefore, I think, be remembered as an additional reason why all excessive squeezing should be avoided in attempts at reduction, and as another argument in favour of early operation. I believe that if, after a fair trial under chloroform, the hernia cannot be reduced, and if, in old and large herniæ, there is no great urgency

after the application of ice or the use of enemata, the protrusion is not speedily replaced, the sooner the operation is performed the better. As to the question of opening the sac, I am of opinion that every effort should be made to avoid doing so, short of returning a hernia, strangulated within its own sac, into the abdomen.'

The following remarks by my fellow-student and friend Dr. Herbert Baillie, upon the *Treatment by Opium of Strangulated Hernia, as it occurs in natives*,¹ are very noteworthy. He observes that, on the occurrence of strangulation in a European, there are few surgeons who would not at once employ the taxis, under chloroform, especially when the strangulation was recent and acute, and, failing in the attempt at reduction, would at once operate. Such at least would be his practice; but, in native practice, he is of opinion, such a method of proceeding would, in many instances, lead to bad results; for, in the case of natives, our aid is seldom invoked until great delay has taken place and much mischief done by the unsuccessful efforts at the taxis of incompetent persons, or of the patient himself, who is often brought to us with the tumour much bruised and injured, although this may not be apparent on account of the dark colour of the skin and the uncomplaining character of the patient. Not being aware of these circumstances in the early part of his Indian career, he generally operated soon after seeing the case, and this gave him several opportunities of examining the condition of the sac and its contents, after they had been subjected to the usual rough handling, and it was seldom that he found the intestine in such a state as would have warranted its return; nothing was left but to divide with the bistoury or tear with the fingers the stricture, and leave nature to repair the mischief caused by meddlesome interference. The sequence of such proceedings was sometimes an artificial anus—than have which most natives would rather die. This happened to an old Brahmin, and nothing could exceed his chagrin at the result of his surgeon's interference, though he did well and lived more than fifteen years after the operation, attempts to remedy the lesion having proved unavailing. These occasional misadventures led Dr. Baillie to resort to the opium treatment which he had seen in vogue at Guy's. The result is shown in the following abstract of the cases of fifty-six men, aged from fourteen to ninety, suffering from strangulated hernia. *Five* were operated upon and recovered; *ten* were operated upon and died; *two* were relieved; *thirty-nine* were treated by full doses of opium and recovered. When he wrote, he seldom found that an operation was called for, the intestine generally releasing itself after the patient had been for a few hours under the full influence of opium. Should the taxis, however, be required, it is then made under the most favourable circumstances;

¹ *I. M. G.*, vol. iv. p. 212.

the parts have been left alone, the patient is composed, and a very little manipulation usually suffices to return the bowel; even then, should the taxis fail, an operation may still be staved off for a time, if no urgent symptoms be present. He is by no means sure that the danger of delay is greater than that incurred by returning into the abdominal cavity a highly inflamed intestine. Indeed, he considered that some of the deaths which followed his operations were the result of peritonitis set up by the presence in the peritoneal sac of the returned portion of bowel. Hence he infers that, instead of the usual rule, *when in doubt, operate*, it would be better (where natives are concerned) to alter it to *operate when not in doubt*, i.e. when all is known regarding the strangulation, that it is recent and acute and has not been tampered with. Then, should the taxis fail under chloroform, and the symptoms be of such urgency as to indicate the operation, operate at once; but, as has been noticed above, such knowledge is rarely to be expected in native practice. He recommends half-drachm doses of Battley's liq. opii sed., repeated every two or three hours till the patient is *fully* narcotised, cold being, if thought necessary, applied simultaneously.

My friends Sir Joseph Fayrer and Brigade Surgeon J. B. Scriven, were among the first in India to employ the modern operations for the radical cure of hernia. The former has recorded, in the above named works, a great body of cases, European and native. I believe that Mr. Scriven's first paper on the subject is that on the 'Radical Cure of Inguinal Hernia,' No. XI., p. 126, of the 'I. Annals.'

In India, *Tapping the Bowel* has repeatedly been employed with success in cases of intestinal obstruction. Mr. A. Tomes¹ treated a Hindoo, *æt.* 40, with a large inguino-scrotal hernia which had remained irreducible for six days, taxis and other means failing. At the setting-in of symptoms of strangulation, there was but moderate distension of the tumour with extreme distension of the abdomen. With a fine aspirator trocar, he punctured first the tumour then the intestine on either side of the median line of the abdomen. Nothing but a little reddish fluid escaped from the hernia, although it was resonant; much flatus escaped from the intestine on the right side, and, on the left, not only air but also twelve ounces of liquid fæces flowed through the needle. The operation was followed by relaxation of the abdomen, cessation of vomiting, and relief from pain; the hernia remained irreducible. Opium was freely given; the man passed an easy night, and the hernia was reduced, without the least difficulty, next morning. There was some little constitutional disturbance, with general tenderness of the abdomen, a little increased near the seats of puncture. Otherwise, the man made a good recovery, and was discharged perfectly well and wearing a truss on the seventh day.

¹ *I. M. G.* for 1882, p. 211.

I do not know whether surgeons in the present day recognise the fact that puncture of the bowel was practised by Peter Lowe, an eminent Scotch surgeon, early in the seventeenth century. Lowe writes as follows at p. 245¹ (in which he describes an operation for the radical cure of hernia), of his 'Whole Art of Chyrurgerie: 'Of the Tumour Inguinal, called Bubonocoele.' 'If by these remedies it reduce not, by reason of the abundance of wind, you shall use five or six punctions in yt part with a needle, as I have often proved it despatcheth the wind, so the intestine doth the more easily reduce; if the excrements impasse the reduction, being hard, you must incise the periton, as is set down by Rasis.'

In India, where goats and sheep are liable to great over-distension of the intestines, puncture is frequently employed.

Peritonitis,

In its ordinary acute forms, occurs in India nearly as it does in Europe; but I think it probable that the comparative rarity of strumous taint among them may give the natives of the plains considerable immunity from its idiopathic attacks.

The prevalence of Splenitis and Hepatitis in India leads to involvement rarely of the splenic peritoneum, and frequently of the hepatic. The discharge of pus into the peritoneal cavity from hepatic abscesses often proves fatal. In poor ill-fed natives, acute general peritonitis may advance in a manner so insidious as to escape recognition during life; at least this has occurred in my own practice. Such cases occur out of India, the unfortunate appearing to begin to die at pleura, pericardium, or peritoneum, as the case may be.

Tubercular Peritonitis is rare in that country. I have seen it in general tuberculosis in the native subject. Prep. 451 in Ewart's Catalogue is: 'Small intestine containing, in the sub-peritoneal areolar tissue, a great number of tubercular deposits, varying in size from a mere visible point or granule to that of a pin's head or millet seed. The same appearances were observed over the mesentery.'

An interesting case has been recorded² in which a *Diffused Abscess of the abdominal parietes simulated Peritonitis* in a native *æt.* 25.

Mr. H. M. Greenhow has related³ a case of 'False Peritonitis,' the 'nervous abdominal pain' of Rigby, perhaps co-existing with some peritonitis, in a nervous hysterical English lady, fifteen days after delivery. The diagnosis, as she made a good recovery, was complicated by the occurrence of a slight attack of phlegmasia dolens in the left thigh and leg, followed by tenderness over the femoral vein on the right side.

In the four years, 1880-83, fifteen European soldiers died of

¹ Third edition, 1834.

² *I. A.*, No. 4, p. 695.

³ *Ibid.* No. v. p. 360.

peritonitis. Three women were admitted in 1880, of whom one died. A fatal case occurred among the women in 1881, and two in 1883 [Hæmo or Pyo-Salpinx?].

Tabes Mesenterica.

In common with other diseases of 'strumous' origin, the mesenteric disease of young persons appears very rarely to take its rise in India. Before I went to that country, daily observation taught me that tabes mesenterica was, among the poor of London, one of the most frequent causes of death in early youth. From the moment at which I landed in Calcutta, I almost absolutely lost sight of it—at least in its English form. My observation, however, was almost entirely confined to the children of officers and natives. Had I looked further, I should have seen more of this disease. Morehead, who was perfectly well acquainted with the diseases prevalent during a long term of years at the large Byculla schools, does not even name this disease, and Webb and Fayrer do not mention its occurrence in the Calcutta schools.

Preps. 106–109, 452–53, in the Calcutta Medical College Museum, display strumous disease of the mesenteric glands; but there is nothing to show, except in Prep. 108, which was from a native adult, a patient of my own, whether they are from Europeans or natives, or whether the disease was indigenous or imported. I contributed 109 without apparently noticing the race. Many years ago, Mr. W. A. Green reported¹ two cases of diseased mesenteric glands in native persons; but, in one, the suppuration which had occurred in the glands was probably ichoræmic, and in the other the disease (apparently malignant) was not tabes mesenterica.

As might be anticipated, the children of soldiers and others have frequently arrived in India the subjects of this disease. It was formerly noticed² that 'children, within the first year of their arrival in India, whether from England, New South Wales, or the Cape of Good Hope—but especially from the latter place—become emaciated, suffer from disordered bowels, and fall into a state of marasmus by which many are cut off. The mesenteric glands are invariably found hypertrophied and the intestines contracted; the liver also is in some cases enlarged.' I am not sure that this was tabes mesenterica. The contemporary accounts of the health of the children (mostly Indo-Britons) at the Madras Orphan Asylums do not show that this type of disease had prevailed there. There is, at present, a great deal of tabes mesenterica among European soldiers' children in India. The returns show, in 1880, 56 admissions with 26 deaths. In the following year, there

¹ *I. M. J.*, vol. ii. p. 130.

² *Madras Medico-Topographical Reports*, Madras, p. 89.

were 43 admissions and 24 deaths. In 1882, there were 31 admissions and 15 deaths. In 1883, the admissions were 32 and the deaths 21. In civil practice, I always had, in my wards, miserably unhealthy and poor men, women, and children of all races, but I never admitted a case of this disease. The comparative immunity of the natives of India from struma finds a striking illustration in the entries for *tabes mesenterica* in the Returns for the four years ending 1883. Thus, among the natives, there were, during the whole term, only 3 prisoners, who died, and 5 native soldiers, of whom 3 died.

In inquiring into the origin of this disease in European children in India, a certain allowance must be made for hereditary taint; but it would be very important indeed and not very difficult, to discover, at least approximately, in how many of these disease originated in England, and in how many cases it occurred in children born in India. I noted the following observation in an old review: ¹ 'About this time' [it is not clear what age of English children born in Bengal is here spoken of] 'blue veins appear on the pale face, and on the sides of the chest and belly; the mesenteric glands become slowly affected in many cases.' This observation was probably made, I think, by a garrison surgeon of Fort William, among the poor barrack children. I never saw or suspected the existence of true *tabes mesenterica* in the child of an officer.

I scarcely feel a doubt that some error in the condition of this system of glands is mainly causative of that dire condition *Morbus Bengalensis* (see *Diarrhœa*, p. 565), which is the most prevalent cause of sickness and death among the agricultural poor of Bengal. But this disease is not mesenteric *tabes* of strumous origin, nor is it any disease of these glands discernible by ordinary inspection. Much as I desired to sift it, I was never in a position to do so. I leave the duty to my successors—no inquiry in science, no problem of national philanthropy more absolutely claims their attention than this does.

Malignant Disease of the Mesenteric Glands rarely occurs in India. Prep. 454 in Ewart's Catalogue is one of 'Scirrhus of the Mesenteric Glands.' I contributed Prep. 278, 'General and extensive medullary cancer of the mesentery and mesenteric glands.' The description of No. 327 is 'An alveolar or colloid cancer of the mesentery, removed from a native female' [by Dr. Cheek of Bancoorah]. 'It consists of a great number of small sacs or cysts filled with a gelatinous, straw-coloured material. The growth occupied the whole of the anterior of the abdomen, pushing up and displacing the intestines. It was fixed on each side, a little below each kidney, by roots about three inches in length, which resembled fine adhesions.'

¹ *I. M. J.* (Finch's), 1842, p. 233.

DISEASES OF THE LIVER.

It is indisputable that, although the literature of Diseases of the Liver, as they occur in India, is extensive and replete with important data, our knowledge of these maladies is far from being well in advance. This is, in a large measure, due to the fact that hepatic physiology has not yet been fully mastered; but I think that the want in question is still more attributable to the circumstance that, in India, where the field of observation is so very extensive, few have acted upon the good example set by Budd, Virchow, Frerichs, and Harley, in making hepatic disease a leading subject in the practical study of a lifetime. More than thirty years ago, having, as Secretary to the Medical Department, the duty of receiving every young medical officer on his first landing in Calcutta, I was in the habit of entreating those who appeared to be well-trained pathologists to adopt the observation of diseases of the liver as a special study, devoting at least ten years to the work. No one, as far as I am aware, followed this advice; and, practically, I was never in a position to take this course myself. I, however, received a striking clinical lesson in hepatic etiology and symptomatology in having the questionable advantage of suffering from liver abscess in the rainy season of 1855; and, when a man addicted to clinical and pathological research, lies upon his back for six weeks, subject to what he regards as a mortal disease, he usually devotes a good deal of thought to that malady, both at the time and afterwards. Consequently, I shall now venture to record my own avowedly imperfect knowledge of the most important of these diseases as they occur in India.

Mechanical Injuries to the Liver.

I have collected six instances in which liver abscess more or less clearly appeared to be *excited* by a fall or blow. In all of these, it appeared nearly certain that there was a tendency to this disease when the men were hurt. Four cases occurred in India. In the fifth, Dr. Sutton's and Dr. Turner's patient, a ship's painter, 'had been in various climates.' There was 'ulceration of the colon of dysenteric type.'¹

That the liver is very tolerant of severe injury, when the large blood vessels escape, is shown by two most interesting cases, observed during the Mutiny, by Mr. Partridge and by Mr. Hare,² in which persons who had been shot through the liver recovered.

The subject of Mr. Partridge's case, General, then Captain —, a

¹ *M. T.*, vol. i. for 1882, p. 369, which see for other cases in which liver abscess was considered to be traumatic.

² *I. A.*, No. xi. p. 164.

very healthy and temperate officer, *æt.* 31, was struck, at the siege of Lucknow, by a musket bullet which entered in the median line immediately below the ensiform cartilage, and, passing backwards, was excised about five inches from the spine on a level with the margin of the tenth rib. He, at first, suffered very severely from irritability of the stomach, the vomited matter being slightly bloody, as Mr. Partridge considered, from sympathetic congestion of the gastric mucous membrane. This, however, yielded to sedatives and salines, and had entirely disappeared previous to his admission to the field hospital on the 13th day. There was a very copious discharge of bile from both wounds, and a small portion of the margin of a rib came away. I saw and carefully examined my friend, Captain —, after he came down to Calcutta. There was a depressed cicatrising spot, adherent to the rib behind, which showed that it was the lower edge of the last floating rib that had been shot away. He told me that, one day, the medical subordinate, who was about to dress his wounds, was astounded at seeing a bright red something protruding, like a finger, from the posterior wound. This, being drawn out, proved to be a portion of the front of his flannel shirt which, happily, was the only garment which covered the pit of his stomach when he was shot. Mr. Partridge and he went home in the same steamer. From the fatigue of the voyage, he suffered slightly from pain in the hypochondrium; but, after landing and enjoying perfect rest, he never had an unfavourable symptom. I watched his healthy and very active life, he being a great walker, during many subsequent years in Calcutta, and I saw him about five years ago, a singularly well-preserved general officer.

Mr. Hare's case is even still more remarkable, seeing that, although the fact did not appear in the official and published report, its subject was, as I understood, a most incorrigible drunkard. A ball entered the liver and passed through it. It lay near the spine. He was very faint; but, by the next morning, seemed to have recovered from the shock, although his pulse was still weak and thready and his countenance anxious. Towards evening, he complained of constant short cough, as if the diaphragm had been touched by the ball in its course. On the evening of this day, there was great prostration, and it was not thought possible that he could live through the night; towards morning, his lower extremities became quite cold, but his pulse was steady. Under the free use of stimulants, he rallied again as day broke. There was temporary constipation, which Mr. Hare was careful not to interfere with; but a dose of castor oil relieved him. His only troublesome symptom was a short, frequent, spasmodic cough from irritation of the diaphragm. Healthy pus escaped from the wound, which could be seen and traced into the substance of the liver. He subsequently had diarrhœa and intermittent fever, to which latter he remained subject when at Simla eight months later. He then had

pain in the liver if he attempted to ride or take strong exercise. This case is rendered the more singular by the fact that, in one living as he did, the liver, thus severely injured, could not have been healthy. It will be noticed that there is no mention, in the above narratives, either of general hepatitis or of jaundice. As a rule, bile is well secreted and jaundice does not occur in liver abscess; and, usually, the liver substance almost immediately outside liver abscesses appears perfectly normal and uninflamed. Although the liver is not a dual organ, nature has endowed it with so vast a system of blood-vessels, cells, and ducts that, when only portions of the gland are injured or diseased, those parts of it which escape lesion, although they may be small, are perfectly competent to perform its functions.

At page 254 of the 'Pathologia Indica,' Dr. Webb has cited a case in which my friend, Dr. John Macpherson, cut off a portion of the liver, of about the size and shape of the four fingers of the hand lying side by side. The patient was an aged Hindoo wounded by a spear thrust. It did not appear that the weapon had touched the liver. A double ligature was placed through the stump and tied on either side after two arterial twigs, which bled very freely, had been taken up. 'For a day or two, the patient was rather low, and had slight irritative fever, and the bowels remained costive. These symptoms yielded to a few purgative doses. In nine days, no bile having exuded from the granulating surface, the ligatures came away with a small slough of liver, and he returned home with the wound healed in three weeks. He appeared, two months later, as prosecutor in his own case. He was in perfect health; there was a little puckering of skin about the wound, and the liver was evidently adherent beneath.' Here will also be found reference to other cases in which portions of the liver, in man and animals, have been excised.

Dr. Waring has shown¹ that 'in London and Bombay, the proportion of deaths from Hepatic Disease, compared with deaths from all diseases, very closely approximates,' the ratio in London being 1 in 59 or 1·59 per cent.; in Bombay, 1 in 54, or 1·54 per cent.

Bilious Flux is described in the chapter on Cholera, page 296.

Defective Excretion of Bile is a frequent attendant upon chronic diarrhoea in India. It has been considered under that head, pp. 564 and 570. At Chittagong, near the sea, the mornings, even during the hot season, are often chilly, with a sea fog. On such mornings, I got quite into the habit of expecting to find nearly every European child in the station attacked with purging, the stools being deficient in bile. Giving some soda in aniseed water, and desiring that the little patients should be kept warm and should have proper food, some mustard oil being rubbed over the hepatic region, I generally found that, as the morning

¹ *I. A.*, No. vi. p. 477.

chill passed off and the sun became powerful, the liver resumed its healthy action before the day was over.

Chronic Congestion of the Liver,

In its simple functional condition, is far more prevalent in cold than in tropical climates—in England than in India. [I do not here speak of congestion of the hepatic venous system in obstructive pulmonary and cardiac disease, which occurs everywhere.] In London, thousands suffer from chronic hepatic congestion, caused mainly by cold and imperfect cutaneous action. Thence result habitual constipation, a sallow countenance, muddy conjunctivæ, and constant dyspepsia. Occasionally, the liver makes a sudden effort to relieve itself, and the result is a '*Bilious Attack*,' during which more or less duodenitis may or may not be present. In the hot climate of India, there is not much of this hepatic dyspepsia and, in the majority of European residents, the bowels act freely. Still, as I have already said, in describing *Bilious Flux*, in the chapter on Cholera, p. 296, moderate hepatic congestion, caused by checked perspiration and good living, often relieves itself, at the end of the Indian cold season, by a sudden and profuse but transient outpouring of bile. My own experience confirms that of Dr. Peet, who noticed that these attacks are rare amongst persons who are acclimatised, and are almost unknown in the natives of India.

I must confess that I know nothing of that mysterious disease called '*Liver*,' from which my medical brethren at home do, or did formerly, think that most old Indians suffer on their return to Europe. I seldom, if ever, had to tell a patient in India, '*Your liver is out of order*,' or, '*You must take something to touch your liver*.' I suspect that much of this notion has come down, traditionally, from old times when the sallowness of persons long resident in India either arose from malarious blood disintegration, or was the result of functional or organic hepatic disease caused by heroic dosing by mercury. Doubtless many a retired Indian, who would have recovered under a course of quinine in his native air, has succumbed to the '*emulgent*' action of blue pill and the Cheltenham waters.

In India, a marked stage of *Active General Hepatic Congestion* often precedes the formation of hepatic abscess. It is extremely needful to detect early this preliminary phase of congestion, because, when we can watch the patient from the first, and can recognise it and treat it promptly, as we often do, it is generally within our power to cut the disease short, averting the occurrence of abscess. In my own very marked case, ending, apparently, in a central liver abscess and inflammation of the peritoneum covering the convex surface of the viscus, the symptoms were as follows. Having been much out of health for some weeks with symptoms which I shall describe when speaking of

liver abscess, I was driving down to my office, as I had been quite able to do every day, when I felt a very distressing, but not acutely painful, sense of weight and tension at and to the right of the epigastrium. Thinking that the cause might be flatulence, although I had never experienced similar distress, I stopped at a druggist's and took a carminative draught containing aromatic spirit. This gave no relief, and, the uneasiness increasing, I was obliged to leave my office in an hour or two and to go home and to bed. Then my sensations were that I felt very ill, depressed and chilly, *but no rigor ushered in the attack*. I believe my condition to have been one of moderate shock, caused by disorder of a vital organ. I, by that time, felt what I have often heard others describe, that which was not so much a pain as a sensation of great and most oppressive tensive weight, as if a six-pound cannon shot (weight) or a half quartern loaf (tension) had quietly taken up its position in my liver.

Although it is unquestionable that very acute congestion generally precedes the formation of liver abscess, examination of the parts after death, in cases where the points of suppuration are small, reveals such an entire absence of inflammatory lesion in the remainder of the organ as to throw absolute doubt upon the existence of such a condition as general hepatitis of the whole viscus. If the liver were liable to inflammation occupying its entire substance, as the lung is, death, from cholæmia, would probably occur in a very few hours after the whole of the cells became involved. I think that my own sensations alone showed that the entire organ was congested; but this state is transient; and, upon examination of small isolated liver abscesses, we are inclined to say 'this condition is no more the result of "*hepatitis*" than a small boil in that locality can be looked upon as the outcome of "*inflammation of the thigh*.'"

Treatment in the Stage of Active Congestion.

Had my knowledge been fuller when I was thus attacked, I should have immediately adopted the following means, which would, doubtless, have removed the congestion and thus have prevented the occurrence of abscess. In the absence of dysentery, a full dose of castor oil should be given at once, and a small one ought to be repeated daily. The application of large sinapisms, repeatedly, over the whole hepatic region. Painting with liquor lyttæ, causing a blister about two inches wide and eight inches long, across the epigastrium, slanting down below the right costal cartilages. This to be kept actively open, by lytta or savin dressing, until recovery becomes complete. Ipecacuan powder (I seldom gave more than from fifteen to twenty grain doses): ipecacuan, thus given, will generally act upon the bowels. Very considerable experience of this treatment leads me to believe that, whenever we can

give it fair play, before suppuration occurs, abscess may generally, nay, in all probability, always be averted.

Assuredly, we have abundant evidence to show that, in hepatic disease generally, ipecacuanha is capable of effecting all the good which mercury was expected to do, and did not do; and that it has never caused any of the evil which, Maclean has shown us, mercury constantly did, especially in liver abscess. Here I may dismiss, in a few words, the subject of the action of mercury in hepatic disorder. For some time before Dr. Maclean, of Netley, effected the vast benefit to suffering humanity in India of denouncing and putting down the destructive employment of mercury in hepatitis and other diseases of the liver,¹ physicians had begun to perceive the irrationality of giving this drug whenever there was 'anything wrong,' no matter what, with the liver.

Thus Dr. Abercrombie wrote, 'In the liver diseases of this country' [England], 'mercury is often used in an indiscriminate manner, and with very undefined notions as to a certain specific influence which it is supposed to exert over all the morbid conditions of this organ. If the liver be supposed to be in a state of torpor, mercury is given to excite it; if in a state of acute inflammation, mercury is given to moderate the inflammation and reduce its action.' Long after Maclean wrote, 'Jog the liver with blue pill' was, in England, an unfailing apothecaries' axiom in every form and degree of 'liver;' and this reckless practice is probably not yet extinct, especially when an old Indian, 'who must have a faulty liver,' is the victim. Nineteen years ago, an elderly lady (not from India), at Cheltenham, told me that she was taking blue pill *every twenty-four hours*, upon the prescription of a doctor who had practised in the East. *Pace* Dr. Maclean, she still lives. Someone has said that to give mercury for 'liver' is to send a madman with a drawn sword into the children's room. I had occasion to recognise this truth vividly in my own illness. After I had suffered much for about a fortnight, I felt so nearly well that I got up, expecting to resume work in a day or two. A dear friend and old fellow-student, who had attended me most sedulously, ordering a taraxacum mixture, &c., which I declined to take,² then said that one small alterative dose of blue pill would complete my recovery by clearing out my liver and rendering its action free. I replied that, mercury acts upon the liver as a cholagogue and excitant, as direct diuretics act upon the kidneys, and that my liver had already endured sufficient irritation, and now demanded rest. When, however, I was told that

¹ *J. A.*, No. iii. for 1854.

² Taraxacum is quite useless as a medicinal agent in hepatic and, as far as I have used it, in all other diseases. I am perhaps to blame for having employed its solid extract in making pills where a grain or less of ipecacuan is given. It, however, does no harm, and the pills remain moist, and readily dissolve.

my conduct was unfriendly, in slighting all advice, I most reluctantly yielded and took a little pill, with the mental proviso 'Surely this *one* cannot do much harm!' Here I was quite wrong; immediately this almost minute dose began to work—and, as I was and am confident, as the result of no other cause whatever—it acted almost as distinctly as the spark of a slow (not very slow) match acts upon gunpowder, renewing and greatly increasing the pain, and again lighting up and aggravating the congestion. I was confined to bed, in a second attack, with hepatic peritonitis, shivering, and sweating; with indeed every symptom, except pointing and discharge, of a large central abscess. I have never witnessed a more crucially demonstrative experiment.

I never bled from the arm or applied leeches or gave mercury in any form in hepatic congestion or 'hepatitis.' Leeching is still advocated by high authority. I think that it and V.S. may give relief, at least temporarily, where there are a sense of tension and great pain in inflammation of the hepatic peritoneum.

The following comment¹ gives a fair idea of the estimation in which V.S., in liver abscess, was held fifty years ago: 'This case is a strong proof of the intractable nature of acute disease of the liver, if not treated early, and by the most steady, decisive, and repeated depletion.' I notice that the patient here 'had appeared for a long time in delicate health.' Still he underwent 'a bleeding of only one pound, after which twelve leeches were applied.' I feel absolutely certain that, had I undergone such depletion, it would inevitably have put me out; but it is clear that the surgeon who employed it was regarded as a sadly indecisive practitioner by his medical seniors.

Waring's case, No. 68, was in hospital $4\frac{1}{2}$ months, in 1830. During that time, he was bled [not by Dr. Waring] four different times, to the amount in all of sixty-eight ounces, and he had on sixty leeches; he took purgatives daily as well as tartar emetic and calomel. Salivation was established eight days after admission, and his mouth continued sore nearly up to the time of his death. In Case 69, the man lost sixty-four ounces of blood by V.S., had sixty leeches, was salivated, and had three blisters, besides issues. In both of these cases, the abscess made its way through the diaphragm and, before they died, there was diarrhœa.

A return of a dragoon regiment in Madras in 1838-39² gives statistics of V.S. and leeching in which it is shown that V.S. to from 90 to 138 ounces was employed in 22 cases with 20 recoveries; and that from 100 to 224 leeches were applied in 78 cases, 10 only of which proved fatal. Some only of these are described as cases of very severe acute hepatitis.

¹ Three cases of abscess of the liver, communicated by the Medical Board of Bengal, *C. M. T.*, vol. v. 1831. p. 427.

² *Madras Quarterly Medical Journal* for March 1839,

Mackinnon held that, in acute congestion of the liver, 'active depletion is necessary and usually very successful.' Macgregor insisted that 'no medical practitioner, at all acquainted with the inflammatory diseases of warm climates, will deny that the lancet is the only weapon we possess capable of checking and subduing Acute Hepatitis.'

Now, many of our patients die, mostly those who are brought for treatment late. But I do not think that we often kill them by heroic treatment. Morehead had faith in ipecacuanha (p. 388), but continued to uphold general and local blood-letting (pp. 385-6); mercurial purgatives (p. 386); and 'the mild constitutional influence of mercury' (p. 389). 'When,' he says, hepatitis, 'in a good diathesis, is at that stage in which the exudation of lymph is likely to be going on, mercurial influence is indicated. If, on the other hand, the diathesis is bad, or there is reason to believe that suppuration has already taken place, mercurial influence is contra-indicated' (p. 389). I commenced my medical studies at a time and at a place where 'STHENIC' disease was not commonly met with—Guy's Hospital in 1835. I have, ever since, been seeking for sthenic idiopathic inflammations—pneumonia, pleurisy, pericarditis, hepatitis, peritonitis, in which, had I met with them, I should, for many years, have been quite willing to bleed; but I have never seen such inflammations, and I did not believe in their existence while and where I practised.¹ In my experience, dangerous idiopathic inflammations invariably occurred in cases where 'the diathesis was bad,' never in a good diathesis. Sthenic cases may possibly have occurred in Morehead's and his predecessors' times.

The ease and the almost certainty with which, by employing the means which I used, we are able to prevent acute congestion or so-called 'Hepatitis' from going into abscess appears to be a strong evidence against the theory that liver abscess is caused by portal phlebitis or embolism. It may be asked 'How does abortive treatment, when it is employed early in the congestive stage, cut short the formation of liver abscess?' I have often found, in my own person, that, by freely incising a boil, on its very first appearance as a small pimple, exit is given to a good deal of blood, and the boil is not developed. See the chapter on Surgical Treatment of Hepatic Abscess, p. 654.

When we have removed that acute 'congestion' or 'hepatitis' the end of which, when it proceeds unchecked, is the formation of abscess, we can, as physicians, do little for our patients except hygienically.

¹ 'I am firmly persuaded by my own observations, and by the records of medicine, that there are waves of time through which the sthenic and asthenic character of disease prevail in succession, and that we are, at present, living in one of its adynamic phases.'—Sir Thomas Watson.

Causes of Hepatic Abscess.

To those who desire to study deeply the most important and interesting but very perplexing question of the causation (there are doubtless more causes than one) of liver abscess, the monograph of my friend Dr. Waring, *An Inquiry into the Statistics and Pathology of some Points connected with Abscess of the Liver, as met with in the East Indies*, 1854, will be found replete with most useful and reliable data. It is based upon the histories of three hundred fatal (chiefly European) cases. The histories of three hundred cases of recovery from the same disease (Waring gives 25) would form a highly valuable sequel to this work.

Some insight into the causation of this disease ought to be afforded by statistics showing (1) its Prevalence in various parts of India, (2) the Race, (3) Age, (4) Sex, (5) Term of Residence in India, and (6) Habits of Life of its victims.

1. LOCALITY.

The following data, for 1883, are from the Sanitary Commissioner's Report :—

HEPATITIS	European Soldiers		Native Soldiers	
	Admissions per Mille of Strength	Percentage of Deaths to Treated	Admissions per Mille of Strength	Percentage of Deaths to Treated
Bengal . . .	26·8	3·93	1·4	10·53
Madras . . .	41·2	4·16	1·0	14·27
Bombay . . .	23·2	3·54	3·1	10·96

In his *Analysis of the Later Medical Returns of European Troops Serving in the Bengal Presidency*,¹ my friend Mr. Hugh Macpherson shows that, as regards *Acute Hepatitis* the admissions, during the eight years ending 1853–54, numbered $3\frac{3}{4}$ per cent. of strength, against $\frac{1}{5}$ per cent. for ten years in England. It caused, in India, $\frac{1}{4}$ per cent. of deaths to strength against $\frac{1}{33\frac{3}{8}}$ per cent. at home; and the deaths to treated were $7\frac{1}{3}$ per cent., and at home only $1\frac{3}{4}$. In other words, the disease was eighteen times more common, carried off seventy-one times as many soldiers, and was four times as fatal to those attacked in Bengal as at home.

From *Chronic Hepatitis* the number treated in Bengal was $7\frac{2}{5}$ per cent. of strength against $\frac{3}{11}$ per cent. in the United Kingdom; the deaths to strength were $\frac{1}{10}$ per cent. against $\frac{1}{50}$; and the deaths to treated $7\frac{3}{10}$ against $8\frac{1}{4}$ per cent.

That is to say, the malady was five times more prevalent, and destroyed five times more men in Bengal, but was slightly less fatal to individuals attacked.

¹ *I. A.*, No. viii. p. 576, and ix. p. 243.

In his recently published monograph on Hepatic Abscess¹ Dr. George Harley cites statistics by Surgeon-General Edward Balfour which show that, as regards *High Altitudes* in India, the three lowest death rates are met with at 1,056 feet (0·46), 2,320 feet (0·59), and 1,830 feet (0·65) respectively. The three highest at² 1,500 feet, 7,800 feet (2·36), 6,941 feet (3·17), and 1,791 feet (4·27). 'In what,' inquires Dr. Harley, 'consists the mysterious factor which so potently rules the mortality of Europeans from hepatitis in India, that even its naturally-supposed-to-be healthy hill stations are so deadly? As the hill stations are of all others those which might have been *à priori* expected to be the most salubrious in regard to this disease (?).' I am not prepared fully to answer this question, but I apprehend that the explanation is not far to seek. Thus these tables show that the decidedly healthy hill sanitarium, Darjeeling, which stands at an altitude of 6,941 feet, has a European mortality of 3·17 per cent. from hepatitis. If the arrangements there are what they were in my time, no European force is maintained, but many soldiers in broken health, fit subjects for liver abscess, are received at the sanitarium. No one wholly blames the genial climate of one of our own southern watering-places for the high mortality rate of phthisis which obtains there. I never heard that old residents at Darjeeling were liable to hepatic abscess. Regarding the present condition of Secunderabad, which has an elevation of 1,791 feet, with a death-rate from hepatitis of 4·27, the *recent* facts at my disposal are incomplete. (It is noteworthy that, in the decennium 1870-79, the death-rate here was 19·9 per mille against that of Madras 12·7, and that of Bellary 9·8. In 1883, the death-rate of European soldiers at Secunderabad was only 8·33, the rate for all India being 10·88.) But those who desire to examine this question will find that the early history of this station, given in the powerful reports of Sir Thomas Crawford, and Dr. Maclean of Netley,³ shows, that, while the hot season is 'very oppressive and exhausting,' there is, in the cool season, 'occasionally a dry, bleak north wind which drinks up the dews of heaven, licks the moisture from the surface of the body, and drives the blood to internal organs.' Of the old infantry barracks it is said 'the mortality in them, chiefly from epidemic dysentery, had been, for years, so shocking as to give them, throughout India, an infamous notoriety.' Of the new barracks it was said, 'Dysentery continued to scourge regiment after regiment quartered in them, with a violence as severe and a mortality as great as in the worst years of the old buildings.' I need hardly say that dysentery and liver abscess generally march together.

Dr. Harley, continuing to cite these valuable tables, observes that,

¹ *Medical Press and Circular*, vol. i. for 1886, pp. 1 to 190.

² There appears to be an error of press here.

³ *Army Medical Department Reports* for 1860, p. 313.

'when the average mortality of all the stations is considered, it gives us a *death-rate from hepatitis of 1.44 in the dry, and 2.65 in the wet season, the idea is suggested that rainfall has possibly some influence on the mortality.*' Dr. Harley's reasoning on this subject will, I am certain, be studied with deep interest in India, especially at the stations which he names. Here we have to reconcile such facts as these (upon which, I apprehend, a medical officer, long posted at all the stations, would be able to explain much). Chittagong, on the eastern sea board, has about 90 inches of rainfall and appears to be exempt from liver abscess. Port Blair, also eastward but far south of Chittagong, has a rainfall (determined by the law which gives Chittagong a heavy rainfall) of 117.39 inches and a death-rate from Hepatitis of 0.83 (is not this a native rate?), while Cannanore, which stands right across the peninsula on the west, but nearly on the parallel of Port Blair, has a mortality of 4.11, with a rainfall of 121.60 inches. Many sicken in the hot season and die in the rains, *vide* p. 623.

The combined influence of Intense Heat and the Paludal Poison have been much insisted upon as causative of liver abscess; and the majority of cases are proved to have been exposed to these influences. On sifting this question, we find (1) as regards *Heat*, that the operation of mere heat cannot be accepted as a cause—bakers and steamship stokers not being especially subject to this disease; (2) as regards the *causative influence of Malaria*, the malaria of temperate climates is not a cause of this lesion. I know nothing to show that those who are 'liver grown' from malarial fever, either in England or in India, are liable to hepatic abscess. In India, the tendency in these cases is to kirrrosis. I have seen two cases in well-conditioned young men who, never having been previously in India, contracted the disease in the voyage round the Cape. Liver abscess is almost or quite unknown in several of the most notorious fever districts of India, while in localities where liver abscess most abounds, as a great part of the Madras Presidency, *vide* the observations on 'Spleen' of Annesley and Day at p. 221, the incidence of marsh fever is less grave than it generally is in the plains of India. When again we are told that hepatic abscess probably depends upon the *combined operation of Heat and Marsh Miasm*, we shall find, in considering, a few pages further on (p. 626), the history of this disease in the West Indies, that here, coincidentally with the fullest influence of these supposed causes, hepatic abscess is not prevalent.

Rapid and great Transitions of Temperature have appeared to be operative probably rather in exciting than in causing this disease. At Bangalore, in the Madras Presidency, there is often so cool a breeze that the soldiers used to expose themselves all day in the blazing sunshine, and, sleeping at night, were visited, when perspiring, by a cold wind from higher ground. Hence it was supposed that the great tendency

to this disease among the troops stationed there arose. I lately heard (not so fully as I could have wished) of the case of an officer, retired after long Indian service, who, standing on the shore at Ryde on a hot day, and seeing some boys bathing, determined to have a dip himself. He got a chill and died of hepatic abscess. Here dysentery may have been set up, or an ancient encysted abscess may have been present; still, taken with the following, this case hints at the possibility that there are quiescent states of the liver (because, if he had felt any uneasiness about the liver, this poor man would probably not have been walking out and disposed to bathe), which a chill may excite into supuration. I have already mentioned [at page 15], the case of a gigantic officer whose distinguished service during the mutiny must have been attended with severe trial by fatigue and exposure, who, when strolling in the hills, and feeling hot, undressed and stood under the fall of a little stream. Suddenly, an agonising pain, which he resembled to the thrust of a bayonet, passed through his liver. He fell in a heap and reached his quarters with great difficulty. The result was an attack of acute hepatitis.

The old theory that, the air of the tropics being rarefied by heat, the lungs have difficulty in obtaining sufficient oxygen, and that, so, an undue share in the function of decarbonising the blood is thrown upon the liver, is specious, but ought to be borne in mind.

2. RACE.

Vide the Sanitary Commissioner's data in the table at the head of this chapter, p. 612.

In his analysis of fatal cases collected from all sources, Waring shows (p. 111) that, out of 300 deaths, only 9 occurred in persons stated to have been born in India. Of these, 6 appear to have been pure natives, either Hindoos or Mussulmans, the others were 'Indo-Britons, or persons born in India of European parents.' Among the general population, the natives of *Bengal* and *Madras* are less subject to liver abscess than Europeans are. Twining considered, from experience in *Bengal*, that 'acute liver disease, terminating in abscess, is exceedingly rare among Asiatics.' In that Presidency, only 9 cases, in natives, came within my personal observation in civil practice.

In *Madras*, Waring gives a table showing that, in the principal jails, during a period of 10 years, out of an aggregate of 51,775 native prisoners, there were only 29 admissions for hepatitis, and 6 deaths.

In *Bombay*, on the other hand, Morehead states that, 'in six years, the deaths from hepatitis, in the Jamsetjee Jejeebhoy Hospital for natives, amounted to 125, and that 55 cases of hepatic abscess in natives, of which he had kept records, formed but a part of those which had come under his observation.'

The S. C. R. show that, in 1883, there occurred, in all the jails of India, only 41 admissions for hepatitis, with 8 deaths.

3. AGE.

Dr. Waring and Dr. Macpherson, and, I think, all other observers, agree that *Childhood*, which is especially liable to dysentery, enjoys an almost complete immunity from idiopathic hepatic abscess. Where this does occur in very early life, it is probably of tubercular origin.

In the four years ending 1883, there occurred among the soldiers' children throughout India, a total of 13 cases of hepatitis, with only one death.

Neither Morehead nor I saw hepatitis or liver abscess in childhood. Morehead showed that the ratio of hepatitis in children was, when he wrote (1860), in Bengal 0·05, and in Madras, 0·3 of strength. He cited the returns of the Byculla schools as proving the rarity of the disease in Bombay at the same time of life; the admissions, in 17 years, were 9, and 1 death in a strength of from 250 to 300 children.

Surgeon-General Hugh Macpherson shows, in his analysis of the returns of European soldiers in the Bengal Presidency for the four years ending 1853,¹ that acute inflammation of the liver brought under treatment only $\frac{1}{20}$ per cent. of soldiers' children, with no death.

I have only been able to find three accounts of cases of hepatic abscess in children. In a child of 17 months;² in a European child of 3 years and 4 months;³ and in a Parsee child of 10 years.⁴

Mr. Blaney had to prepare the statistics of diseases treated at the Bombay European General Hospital, published by Dr. Stovell, and also those published by Dr. Morehead. He was unable to find a single case of hepatitis in a child.

The ages at which Waring noted the occurrence of liver abscess were as follows, in a total of 227 cases. From 16 to 20 years, 10; 21 to 25, 59; 26 to 30, 53; 31 to 35, 42; 36 to 40, 35; 41 to 45, 13; 46 to 50, 9; 51 to 55, 2; 56 to 70, 3; 72 years, 1.

Macpherson's table xvi. shows that, while hepatitis rarely kills soldiers under twenty years of age, it is very fatal between 25 and 35, but especially between 30 and 35. He found that these inferences regarding soldiers were completely borne out as to the more mixed class of patients treated in the Calcutta General Hospital. A careful analysis of the records made by Dr. John Macpherson, when assistant-surgeon to that institution, led to the conclusion that hepatitis is decidedly uncommon under 20; that it is most common from 25 to 35; and that it is most fatal from 30 to 35.

¹ *I. A.*, No. ix, p. 248.

² *Bombay Med. and Phys. Trans.* for 1861, p. 47.

³ *Ibid.* for 1869, p. 9.

⁴ *Ibid.* No. x, p. 303.

4. SEX.

Waring found that nine *Women* fell victims to liver abscess out of a total of 300 cases of both sexes. Of these women three were born in India. In his invaluable statistical *Notes on Some of the Diseases of India*,¹ Dr Waring further shows that, out of a strength of 6,559, the women of the Madras European army (not all of whom were probably of pure European blood) had, in ten years, 13 deaths from hepatitis—which was at the rate of 0·19 per cent., the ratio to strength of private soldiers being 0·68, and of officers 0·34.

I treated only one case, which proved fatal, in a native woman; but I have heard of a few cases of liver abscess in which well-to-do European ladies died of hepatic abscess.

According to the S. C. Reports for the four years ending 1883, the admissions for hepatitis and deaths of European women in all India were: 76 and 4; 61 and 3; 45 and 5; 51 and 3.

Macpherson's analysis, cited above, shows that, in the four years ending 1853, the barrack women in the Bengal Presidency suffered from acute hepatitis at the rate of $1\frac{3}{5}$ per cent. Of these cases $4\frac{1}{2}$ proved fatal.

5. TERM OF RESIDENCE IN INDIA.

We have abundant statistics with regard to this point. Waring gives an analysis of 114 fatal cases of liver abscess, from which he shows that nearly 23 per cent. died under one year's residence, and more than 50 per cent. within the first four years. A larger number died in the first year than during the ten years between ten and twenty. 'Length of residence in India,' he observes, 'seems to confer a great immunity from suppuration of the liver; and, when it does occur in those who have been long in India, it is almost invariably associated with irregular and intemperate habits.'

Hugh Macpherson gives a table (xvii.) which shows that, among our European troops in Bengal during the four years ending 1853-54, the number of men who died under 1 year's service was 8; 1 to 3 years, 63; 3 to 5, 55; 5 to 7, 45; 7 to 10, 57; 10 to 14, 32; 14 to 20, 2; above 20, none.

6. HABITS OF LIFE.

I have cited above Waring's observation that, when liver abscess occurs in those who have been long in India, it is almost invariably associated with irregular and intemperate habits. In a subsequent report,² he gives a table, from rather limited data, which shows that

¹ *I. A.*, No. vi. p. 481.

² *Ibid.* p. 484.

'double the proportionate number were admitted of those who were known to be intemperate over those who are either teetotallers or persons of known temperate habits.' See 'Prophylaxis of Liver Abscess,' p. 648.

Having shown that children are very exempt from liver abscess (though not to so great a degree as the native), Dr. Waring suggests that we should be led to infer that those conditions or circumstances which cause the exemption in the one case may operate in the other. In what respects, then, he asks, do the conditions of the native of India and the European child approximate? They closely resemble each other in three respects. 1st, lightness and an unrestrained style of clothing (thus allowing unchecked operation to the cutaneous function); 2nd, in simplicity of diet; and 3rd, in the practice of temperance. He considers that, in the direct ratio as a person in India departs from either of these conditions, especially the third, temperance, so are the chances of his becoming affected with hepatic disease increased. Next to the native and the child, we see that women are exempt from hepatic disease. They often depart from the above conditions, particularly as far as dress and temperance are concerned, but rarely, if ever, to so great an extent as the officer and private soldier who, for the most part, lead a life as diametrically opposed to that of the child and native as it is well possible to conceive; these are subject to hepatitis in an extreme degree. Rigid temperance *per se* is, however, no absolute preservative against the invasion of disease of the liver in India.

While admitting that the drunkard is more subject to disease than the temperate man is, Morehead considers that the evidence that intemperance in drinking exercises a peculiar influence in causing hepatitis is by no means conclusive (p. 363). Bearing in mind the fact that, during the last forty years, the manner of living, especially as regards temperance, of Indian officers, has generally become as worthy of imitation as that of any gentlemen in the world, and that gluttony and drunkenness are far less common among the private soldiers than they formerly were, I cannot bring myself to believe for a moment that these vices are the *prime* cause of liver abscess in our Indian army, until it shall be explained to me why our English troops in the West Indies, who live under the same parallels, and are, doubtless, as little and as much 'generous livers' as their brethren in the East are, should be almost wholly exempt from hepatic abscess.

The view that, shall we say hypernutrition and stimulation, are largely influential in the causation of liver abscess in India, finds a powerful advocate in Dr. George Harley.

At page 21, I have cited the opinion, on this subject, which I published in 1851. If I were now engaged in re-casting the work in which it appeared, I should adhere to this view unless I could be

certain, as I am not, that all people who use but very little active exercise in India, strictly avoid hot meat breakfasts and hot meat tiffins, and, in regard to dinner, content themselves (as the large majority of gentlemen there have long done) with a hearty plain meal and a little claret or Bass's beer.

Pathological Relations.—As I have already observed [page 311] that, when a person with grave organic renal disease is attacked with cholera, death appears to be almost certain; so, probably, it is that, when abscess takes possession of a chronically diseased liver, repair is nearly impossible. Still, out of 132 cases in which the condition of the liver generally was shown, Dr. Waring found that there were only two in which there was kirrrosis, and three others in which there was evidence of old disease.

Morehead remarked (p. 424) that 'it is generally stated that cirrhosis of the liver and hepatic abscess do not occur together.' 'This,' he considered, 'is doubtless correct of European countries, but it is not so in India, where the co-existence of the affections is not very unusual.' He quotes four cases illustrative of this remark. It is not, I think, maintained by anyone that a drunkard's liver, or a liver rendered kirrhotic in paludal cachexia, has any immunity from suppurative disease. The contention is that abscess is not one of the natural outcomes of either kind of kirrrosis.

In London, hepatic abscess is certainly not a recognised sequence of kirrrosis. Dr. Wilks, whose experience of chronic liver disease is vast, considers that idiopathic hepatitis, ending in abscess, is 'perhaps never seen in this country.' Dr. Thomas Addison used to mention a solitary case (which was perhaps tubercular) in a lawyer who caught cold, having fallen asleep in his chambers. I, in twelve years' steady experience in the *post-mortem* room at Guy's, never saw idiopathic liver abscess—not of hydatid origin—contracted in England, in a hob-nail, nutmeg, fatty, or healthy liver. Here we are met with a striking observation by Dr. Harley,¹ who adduces figures to show that, out of a total of 26,156 native patients treated in the Bombay hospitals, 0·58 per cent. laboured under hepatic abscess; and, of 4,235 Europeans, 0·66 per cent., that is to say, only 0·08 per cent. more; while Dr. Norman Moore gives statistics of 2,464 autopsies at St. Bartholomew's Hospital, London, in 20 of which abscess of the liver was met with, thus showing that no less than 0·81 per cent. of Europeans, dying in temperate England, suffer from abscess of the liver in one form or in another—that is to say, 0·23 per cent. more than the natives of India, and 0·15 per cent. more than Englishmen residing in India. I must deferentially suggest that, before the parallelism of these two examples can be admitted, the precise nature of each of Dr. Moore's twenty cases will have to be considered. Were any of these

¹ *Op. cit.*, p. 117.

cases of tubercular or hydatid disease?—conditions which are practically non-existent in India. Again, how many of these twenty patients brought the disease with them from India and elsewhere? It is to be recollected that, in the old time alluded to above, when I was a young observer at Guy's, the voyage from India occupying, at the shortest, ninety days, very few Indian cases of abscess were likely to reach the hospitals of London. It would be well to ascertain the statistics of idiopathic liver abscess at an inland city—say Birmingham.

Budd's doctrine that liver abscess depends upon *Ichoræmic Absorption* from dysenteric or other ulcers in the intestinal canal, stomach, or gall bladder, or from abscesses or suppurating wounds situated elsewhere, as about the rectum and pelvis, and communicating with portal veins, is well known, and carries more and more conviction with it as observation advances.

At present, the balance of opinion is in favour of Budd's doctrine where *multiple* liver abscesses are coincident with suppurative action within the domain of portal vessels—especially when abscesses are found in other organs—abscesses of the liver and brain, apparently thus originating, occur in enteric fever.¹ Waring has given a very elaborate array of statistics illustrative of the coincidence of intestinal ulceration, chiefly dysenteric, with liver abscess. He shows that, 'out of a certain number of hepatic abscesses, ulceration of the large intestine' . . . 'was present in 72 per cent.' Waring mentions that, of 61 fatal cases of dysentery, dissected by Dr. John Wilson in China, hepatic abscess was found in two instances only. Upon this we may remark that, in many cases of dysentery, especially when, as happened in China, there is a scorbutic taint, the dysentery kills so rapidly that hepatic abscess has not time to form. The observation of Dr. Baly is cited. Out of many hundred cases of dysentery examined by him at the Millbank Penitentiary (1840–47), not one was complicated with hepatic abscess; but, 'in the epidemic dysentery which prevailed in Dublin, in 1818, Dr. Cheyne met with hepatic abscess in 4 cases, out of 30 bodies examined. In opposing Budd's theory, Dr. Waring gives a table which shows that 'out of 2,758 cases of dysentery treated in the Madras Presidency, abscess of the liver occurred 68 times.' But we must set, against these 2,758 cases of dysentery *treated*, Waring's 72 per cent. of instances of intestinal ulceration found in a certain number of *fatal* cases of liver abscess, recollecting that a very large proportion of dysentery cases recover. Did space admit, I could multiply the data adduced by Indian statisticians to show the *proportion of cases of dysentery in which liver abscess occurs*.² Thus Dr.

¹ For an interesting *résumé* of the literature of these cases, *vide M. T.*, vol. i. or 1883, pp. 16 and 100.

² Surgeon-General Moore, *Bombay Med. and Phys. Trans.*, and Surgeon-General Rankin, *Madras Quar. Med. Jour.*, July 1863.

Moore has drawn up a table which shows that the figures of eight observers give, in 1,532 cases of dysentery, only 295 examples of liver abscess, or 18 per cent. I think that Budd's theory of septicæmia is best checked by statistics of *cases of liver abscess in which dysentery was not present*. In 150 fatal cases, Mr. Rankin found:—

Dysentery without hepatic abscess	99 cases.
Dysentery with hepatic abscess	41 „
Hepatic abscess without dysentery	10 „

More cases of abscess without any evidence of dysentery are still needful in illustration of this question: here, of course, it must be borne in mind that the ulcers may heal before the abscesses kill; still, the history of the case ought generally to leave this point clear.

Placing aside tubercular and hydatid disease, there appear to be cases of hepatic abscess in which there is no evidence whatever of ichoræmia. To this Budd might have replied: 'You have overlooked that evidence in your cases, and infer that it did not exist.' Still, there are cases of liver abscess in which no evidence of portal infection is discoverable. With Budd's doctrine always before me, I have persuaded myself that I have treated some of these—such as that of a young officer of remarkable vigour and good constitution, without intestinal disorder, with acute hepatic congestion, in whom I am confident that I averted liver abscess, by counter-irritation, low diet, and a daily dose of castor oil.

Counter-irritation and ipecacuanha assuredly have not the power of counteracting portal septicæmia, embolism of vessels, and necrosis of liver tissue where they have occurred; but I am convinced that such infection of the portal blood does not generally take place where ipecacuanha is promptly employed in dysentery.

An association has been traced¹ between SCORBUTUS and hepatitis; and, doubtless, this has a prominent place among the causes. In commenting upon 21 *fatal* cases of liver abscess which occurred in H.M.'s 6th Foot at Bombay, Dr. Jackson observed: 'Some of the patients evinced symptoms of scorbutus; and I suspect that, in all of them, a latent scorbutic diathesis existed.'

The remarks of Dr. Kinloch W. Kirk,² on the use of limes in hepatic and splenic enlargement, are still well deserving of attentive study, especially by those who have to treat liver disease in districts where scorbutus is prevalent.

It has not, I believe, been considered that abscess is a natural termination of extreme FATTY DEGENERATION of the liver; but much weight must be given to the opinion that fatty change is among the predisposing causes of hepatic abscess.

¹ *I. M. J.*, N. S. iii. p. 411.

² As cited in Webb's *Pathologia Indica*, p. *269.

In 1855¹ Mr. Charles N. Macnamara wrote : ‘ It seems to me very probable that, when the liver is in this state of fatty degeneration, its vitality must be very much lowered, it is therefore more subject to inflammation, and less able to resist its effects ; and thus we may, I think, account in part for the frequent occurrence of abscess in this organ. In several cases in which there has been the most extensive fatty degeneration of the liver, I have found one or more abscesses present.’ I have already cited, at pp. 21 and 618, the opinion on this subject which I published in 1851.

In 1869,² Mr. Dymock narrated the case of a man, *æt.* 42, with a fatty liver, who died, when he had been in India only twenty-one days from extensive liver abscess. He observed, in a second paper,³ that the *post-mortem* appearances noticed where abscess forms in a fatty liver are peculiar ; in the first stage, a portion of the gland appears disintegrated, although still consistent ; it is like a rotten spot in an apple, of a dull drab colour, surrounded or not by a hardly perceptible zone of congestion ; there is no trace of a fibrinous boundary ; at one spot, generally on the surface, liquefaction may have commenced. Under the microscope, no trace of cell structure can be seen ; the field is occupied by finely granular matter, and numerous large and small oil globules, intersected here and there by bands of cellular tissue. In the more advanced stage, and when liquefaction has taken place, the contents of an abscess are very fluid and loaded with granular matter and oil ; near its boundaries, which are ragged, the disintegrated hepatic tissues above described are seen gradually merging into living tissue. We are not told whether there is general fatty degeneration of the liver in these cases. My own observation inclines me rather to consider that the fatty change in these cases is local, and that it may be considered rather as an accompaniment than as a cause of the suppurative mischief.

It was, I think, the late Dr. Edward Goodeve who traced a remarkable resemblance, both in their mode of origin and in their course, between liver abscess and *FURUNCULUS*. In India, boils become prevalent in certain years, as if epidemically. The seasons in which they occur there are the very hottest. Here, as a result of long-continued and inordinate sweating, the skin is overwrought and portions of areolar tissue become necrosed, say by pigmentary occlusion of the arterioles supplying them. In like manner, long-continued intestinal flux overworks the liver, bowel irritation, in its reflex operation on the liver, incessantly urging that organ to inordinate excretive action [Edward Goodeve], and thus necrosis of more or less liver tissue results. Certainly, in India, the season for boils is also

¹ *I. A.*, No. v., ‘ On Fatty Degeneration of the Liver, &c.,’ p. 184.

² *Bombay Med. and Phys. Trans.* for the same year, p. xx.

³ *I. M. G.*, vol. i. p. 298.

specially the season for liver abscess. This explanation accorded with the circumstances of my own case. I was much out of health and liable to attacks of Chittagong fever and neurotic asthma. I had lately entered upon a very responsible appointment, as Secretary to the Medical Board, the duties of which were quite new to me. At the hottest season of the year I had to cross the Hooghly twice daily; the transit occupied about an hour each way, and entailed great exposure. All my scanty leisure was occupied in preparing a large book by order of the Government. I became, in the hot season, the subject of bilious diarrhoea, the stools being always very dark and frequently appearing to be mere bile. Loss of appetite and strength resulted, still I felt equal to my work. This went on for nearly two months into the rains, in which the weather was chilly. In this manner, in innumerable cases, dysentery in the hot season ends in liver abscess at the setting in of the rains, thus leading to the natural but absolute mistake of considering that the season of the rains is the unhealthiest in the year. The fact being that, while the atmospheric coolness of the rains refreshes the healthy, it chills and often destroys those in whom the previous extreme heat has caused disease. One day, in place of the diarrhoea, I got dysentery, which a dose of laudanum and castor oil stopped in twelve hours. A day or two afterwards, I was attacked with liver congestion in the manner described above.

To revert to what may be called the furuncular theory. It may be asked—Why do young European children in India, who suffer most severely from boils and from bowel complaints, enjoy such remarkable immunity from liver abscess? A remark¹ by Mr. Blaney has an interesting bearing upon this question. A child, *æt.* 3 years and 4 months, of European parentage, having suffered greatly from boils, was attacked with liver abscess. This having been opened with a straight bistoury, recovery followed. Mr. Blaney observed, ‘the previous history of the case seemed to show at least a remarkable coincidence between the dispersion of a crop of boils and the enlargement of the liver.’ ‘Previous to the occurrence of this, there may have been one or two discharging boils in that condition which is looked upon as favourable to the decomposition of pus and the absorption of its putrid matter into the system.’ I think that this question ought to receive further attention.

The occurrence of *Lumbrici* in liver abscesses, which has rather frequently been noticed in India, is probably unimportant, because, in the moribund, these parasites generally try to escape, and have, here, in all likelihood made their way by the common duct. Preparation 540 in the museum of the Calcutta Medical College is Liver of a domestic rat (*mus decumanus*), with a cyst in its centre containing *atricocephalus dispar*, which is appended to it. There are also two

¹ *Bombay Med. and Phys. Trans.*, 1869, p. ix.

other cysts containing remnants of similar dead worms.¹ The discovery of *dracunculus* in liver abscess [Morehead and Peet] is a much more important circumstance.

I was much in the habit of seeing *Hydatid Disease* of the liver in England; but, in India, I never met with a case of abscess of the liver in which there was any ground for considering that the lesion was due to the presence of hydatids.

As far as I can ascertain, our only evidence that hydatid disease can originate in India rests upon the debatable case, cited at page 29, of a low caste woman of Bhopal who died with hydatids in the spleen. Hydatid disease was seen by Dr. Garden² in the liver of a man who had resided for eight years in India; and by Dr. Lyons in a soldier's liver. This man had also served for eight years in that country; but it is well known that it is a habit of these parasites to remain quiescent in the liver for many years. Prep. 334 in the Catalogue of the Calcutta Medical College Museum is described as 'two acephalocysts from the spleen of a patient in the Native Hospital.' There is no history of this case; but, as many seafaring natives are treated in that hospital, it is by no means certain that the disease originated in India.

Morehead says that liver hydatids 'are rare in India.' He saw one case in a hussar *æt.* 22, and two cases in natives: in both of these echinococci were present. There is no life history of one of these latter. The subject of the other case was a negro sailor, a native of Muscat.

There is a case by Mr. Thomas Lowe, medical officer, Sappers and Miners, Madras,³ in which a woman (race not stated but probably European) had an abscess of the liver discharged by the bowels, upon which the tumour almost disappeared. The evacuations were found to be 'made up of puriform liquid full of floating hydatids, tinged with bile. Echinococci were very numerous, and not only solitary, but in small globular masses; there were also many opaque yellowish masses of granules of an irregular form. The last evacuation appeared like minute fish spawn.' Under the microscope, these were found to be echinococci. After this, the patient made a rapid convalescence and took a sea-voyage. The reporter observed, 'How long this patient had been the subject of hydatids, it is difficult to say. The sac which contained these acephalocysts had probably existed a long time, and it is possible that the hydatid tumour might have gone on developing, as a pure and simple hydatid growth, for years without even much injury to health.' In this case, 'the hydatid tumour suppurated, and its contents were tinged with bile.' There is no evidence to show that the disease was contracted in India.

I, some years ago, drew attention to a plan of treatment which

¹ Webb's *Pathologia Indica*, No. 185, p. 257, and *Ewart's Catalogue*, p. 125.

² *I. M. G.*, April 1872, p. 85.

³ *Annals of Military and Naval Surgery*, vol. i. p. 216.

may be deserving of notice, especially since hydatid disease of the liver appears to have become, in Australia, a more prevalent and destructive evil than hepatic abscess is in India. I have repeatedly seen, in London, parent cysts, in the liver which had undergone cretaceous change and had evidently become considerably reduced from their original size, filled with dead acephalocysts, compressed into a mass which is deeply bile-tinged. As the accidental opening of a bile duct has evidently killed the hydatids, by the admission of bile, and thus has arrested and cured the disease, it appears advisable that, as soon as a non-suppurating hydatid tumour of the liver is detected, we should inject it with ox-gall. This might be done, through the long canula of a fine exploratory trocar, without risk of effusion into the peritoneum. The danger of employing this measure would, I think, scarcely be so great as that attending the operations for hepatotomy which have of late years been practised.¹ As the use of aspiration in this disease is sometimes successful,² it probably is so in consequence of wound of a duct and admission of bile to the sac.

Having proved³ that *tænia echinococcus* very frequently occurs in cattle and sheep slaughtered for food in the Punjaub, Dr. James Cleghorn has endeavoured 'to show that a great number of the hepatic abscesses that occur in India may be due to suppurative changes taking place in hydatid cysts previously existing in the liver.' Dr. Cleghorn supports his argument very ably, and his reasoning well deserves the attention of Indian pathologists: still I am quite unable to concur in his opinion. Thus he cites the statements of Frerichs, Morehead, and other authorities, to the effect that, in India, hydatids rarely occur in man. I, and doubtless the large majority of other Indian observers, were perfectly familiar with hydatid disease of the liver as it prevails in London. Yet none of us have remarked any resemblance whatever between this condition and the ordinary hepatic abscess of India. No one has demonstrated the presence of hooklets in the pus of such an abscess. And, I repeat, no one has given a case in which it was indisputable that hydatid disease of the liver originated in India. For further details of Dr. Cleghorn's researches, *vide* the chapter on *Parasites*—*Tænia echinococcus*, p. 27.

As abscess of the liver *occurs in the lower animals*—dog, rat, orang-outang, kangaroo, and python, further investigation of such cases might throw new light upon the genesis of hepatic abscess in man; but the rat suffers from tuberculosis in all climates. Webb describes two

¹ Reports of two successful cases by Mr. Lawson Tait, read before the Medico-Chirurgical Society, May 24, 1882, and two others, one of which was successful, by Dr. J. Knowsley Thornton, *M. T.*, vol. i. for 1883, p. 89.

² Mr. Morris in two cases, once twice over. Medico-Chirurgical Society, May 24, 1882. Dr. Bruce, one case, *M. T.*, vol. i. for 1883, p. 609.

³ *I. M. G.*, vol. vi. p. 41. 'Hydatid Cysts and Tropical Abscess.'

specimens, in the Calcutta Museum, of livers of domestic rats 'affected with strumous abscesses,' 'a disease which,' he adds, 'rats infesting Calcutta seem to be sorely afflicted with.'¹ It was long ago observed that the rats of the Paris sewers are for the most part tubercular. Prep. 524 in the Calcutta Museum is 'Liver of a dog affected with a pulpy scrofulous deposit.' Tuberculosis appears to be the most common and natural cause of death in wild animals when in captivity, especially monkeys. Mr. Sutton brought before the Pathological Society, on December 19, 1882,² the liver of a kangaroo showing 200 abscesses on the surface or in its substance. Their walls were surrounded by hard palish material, and they contained pus; one had burst on the surface, causing a fatal peritonitis. He remarked that abscesses of the liver are common in birds. A large python, seven feet long, died four days after its admission into the Zoological Gardens, so that nothing was known of its clinical history. He found a large abscess in the neighbourhood of the gall bladder, containing a pint of pus; the liver contained multitudes of minute abscesses. There was acute pericarditis. The report is headed 'Pyæmia.'

Annesley observed that cases of hepatic abscess are 'commonly met with amongst the fair-complexioned, the scrofulous, the relaxed and leuco-phlegmatic, the enervated, those subject to bowel complaints, and the sedentary. Kenneth Mackinnon remarked that those of fair complexion and light fibre (the scrofulous) are most liable to be affected with hepatic abscess. This agrees with the result of Joseph Ewart's Indian experience, that those who would probably have fallen victims to phthisis, if they had remained at home, are very liable to fatal dysentery in the East.

The *History of Hepatitis in the West Indies* forms a very interesting epidemiological study.

In his treatise on the diseases most frequent in the West Indies, and herein more particularly of those which occur in Barbadoes, published in 1726, Richard Towne gave a very able sketch of the prevalent diseases, including fevers, 'a diarrhœa,' 'a dysentery,' and 'the jaundice,' but does not seem to have been acquainted with liver abscess. Seeing that he does not appear to have made *p.-m.* examinations, this is not very conclusive. Dr. John Hunter, F.R.S., F.R.C.P., and Physician to the Army, says, in his 'Observations on the Diseases of the Army in Jamaica,' published in 1796, at p. 337, that 'It is a remarkable thing that, in the East Indies, under the same latitude nearly as Jamaica, that is, at Madras and Bombay, the disease known in those countries by the name of the *Liver* or *Hepatitis*, should be the most prevailing disorder among Europeans, and that *the same should not be known in the island of Jamaica.*' [Italicised N.C.] 'The

¹ *Pathologia Indica*, p. *258.

² *M. T.*, December 30, 1882.

Liver or *Hepatitis* of the East is an acute disease attacking often suddenly, and soon proving fatal, unless proper remedies be used against it. By its natural progress, it hastens to suppuration, and, if the disease arrive at that stage, there are few to whom it does not prove fatal. I have taken notice of the enlargements of the liver and spleen which occur in Jamaica after long-continued intermittents, the same that are to be met with in all countries in which intermittent fevers prevail; but they are totally different from hepatitis, having no tendency to suppuration, and generally ending in dropsy. I never saw them except as the effects of preceding intermittent or remittent fevers; but the hepatitis comes without any previous disease. There is pain and often fulness in the right hypochondrium, a pain on the top of the right shoulder, and fever. These symptoms have a rapid progress to suppuration in the substance of the liver.' Dr. Hunter made and has recorded a great number of careful *post-mortem* examinations. I have cited the above passage at length, as affording evidence of the fact that he looked in vain for hepatic abscess in the West Indies, 'from the beginning,' to use his own words, 'of the year 1781 till the month of May 1783.'

Dr. Colin Chisholm, F.R.S., who had been Inspector-General of Ordnance Hospitals in the West Indies from 1784, writes (p. 68) as follows in his 'Manual of the Climate and Diseases of Tropical Climates,' under the heading of Acute Hepatitis: 'It is certain, however extraordinary and remarkable it may be thought, that acute hepatitis was scarcely known, as an idiopathic disease, in almost all the West India islands, till about the year 1770. The causes of the disease have always existed, but such was the prepossession that every disease in which febrile heat, a quick hard pulse, and yellow suffusion of the skin were observed, must be bilious fever, an expression to which no definite idea was attached, that further inquiry was deemed unnecessary, and anatomical investigation never resorted to.' [Here it is to be noticed that this description is not applicable to the manner in which medical research was conducted in Jamaica some ten to thirteen years later, between 1781 and 83, when Dr. Hunter collected the materials for his very thoughtful work, and failed to discover that hepatitis and liver abscess had ever prevailed in Jamaica.] 'In fact, when I first settled in practice in that country, I have known the most marked symptoms of hepatic inflammation entirely overlooked; and, whilst local inflammation was thus the real disease, putrid diathesis was alone dreaded, and alone provided against by bark, wine, and other stimulating tonics. The natural consequence followed. Hepatic gangrene, hepatic abscess, and hepatic diarrhoea, have destroyed thousands; and yet improper treatment of the original disease had never been considered as the cause. The disease was imperfectly known and little understood when I first settled in Grenada in 1784' [Dr. Hunter practised in the

West Indies only up to 1788]. Although we find Dr. Chisholm speaking thus vaguely of the vast destructiveness of the gravest hepatic diseases, he only expressly notices one case of hepatic abscess, which was not fatal; and, although he alludes to 'the morbid changes observed after death,' he does not give the *p.-m.* appearances in a single case. He makes the following interesting remark. 'Both anomalous and acute regular hepatitis, accompanied an epidemic catarrh or influenza, which prevailed generally and very fatally in the West Indies in the last month of 1789 and the first of 1790; and, in this instance, the peculiar state of the weather seemed to be the exciting cause of hepatic inflammation. During those months, particularly December, the coldness of the N.E. and N.W. winds was exceedingly piercing, communicating a sensation similar to that which is felt at the commencement of a paroxysm of a regular intermittent.' 'The danger was most pressing.' If my reader will carefully peruse this author's remarks in full, pp. 68-71, he will, I think, scarcely be able to avoid the conclusion that, although Dr. Chisholm fails to give a history of the disease with sufficient detail, hepatitis, terminating in abscess, was very prevalent in the West Indies for some time after 1784.

Those who may desire to inquire fully into this question will find an interesting discussion upon it in Dr. James Johnson's 'Influence of Tropical Climates on European Constitutions,' 1821, under the heading 'Hepatic Derangements.' See also Sir Ranald Martin on 'Causes of Hepatic Diseases in the East Indies.'¹ All observation tends to prove that, of late, liver abscess has not been prevalent in the West Indies. Sir Joseph Fayrer gives² a table which shows that, during the three years 1870-72, in a force numbering from 966 to 1,432 stationed in those islands, there occurred only sixteen cases of 'hepatitis,' and one of liver abscess. Two of these patients were invalided, but there was no death.

In the Army Medical Report of the European soldiers, averaging 879 strong, stationed in the West Indies in 1881, I do not find any mention of hepatitis or liver abscess.

Here one of those who place their faith exclusively upon the septicæmia theory may possibly say, 'In this Army Medical Report of the European soldiers, averaging 897 strong, in the West Indies in 1881, there is no mention of hepatitis, liver abscess, or dysentery; how can liver abscess occur where dysentery does not exist?' I reply, this is merely an illustration of the wave doctrine of disease, or at all events of the fact that there, as probably everywhere else, the prevailing disease of one epoch may be rare or entirely absent at another. Thus, writing in 1726, on the diseases of the West Indies, Richard Towne gives a chapter to 'A Dysentery,' with its Ipecacuanha Treat-

¹ *Lancet*, May 30, 1863.

² *Tropical Dysentery, Diarrhœa, &c.*, p. 202.

ment, and another on the jaundice ; but, as I have already mentioned, does not allude to hepatitis or liver abscess. Dr. John Hunter, writing in 1796, says (p. 337) that, ' the liver or hepatitis is not known in the Island of Jamaica ; ' but that (page 12), ' the disorders that prove fatal to soldiers and Europeans in general in the West Indies are of two kinds, namely, fevers and fluxes.' The dysentery, as it appears in the Island of Jamaica, being ' the same disease that was epidemic in London in the summer and autumn of the years 1779 and 1780 ' (p. 173). Chisholm describes idiopathic and hepatic dysentery as prevailing there in and after 1779. My father was, for about two years, flag-surgeon to the naval Commander-in-Chief on the West India Station eleven years after Hunter came home, cir. 1794. He told me that he saw a great deal of dysentery, but I cannot recall any allusion to hepatic abscess.

Here lies before us a question of singular difficulty. Like India, the West Indies appear to be subject to all the conditions hitherto regarded as causative of liver abscess. These, our Eastern and Western possessions, lie in the same relation to the equator. The two localities suffer in common from heat, rarefied air, rapid and great transitions of temperature, damp, marsh influence and resulting fevers, dysentery, diarrhoea, and both have always numbered too many dissolute, indolent, gluttonous, and intemperate people among their inhabitants. In India, hepatic abscess is always present. In the West Indies, it has occurred, but its appearance there is so uncertain that physicians who have practised there have held that these islands are exempt from it. Hence we must, I think, conclude that the idiopathic liver abscess of hot climates is not essentially due to any hitherto recognised primary cause, and that we have still to search diligently for its true factor—say in some variable or fixed telluric or atmospheric influence, some exhalation, some attribute of volcanic action. I would not for a moment suggest that there is adequate evidence upon which to base the theory that the inhabitants of a volcanic locality in the tropics must be expected to be exempt from hepatic abscess ; but (merely as a hint that, in the existence of a doubt like that which we are now discussing, we must review all known facts that may possibly aid us, and then explore the unknown for new data) I may mention that, at Chittagong, in Eastern Bengal, lying between 23 and 24 degrees of North latitude, where I practised, as the only European medical man, for three years and a half, I never saw or heard of a case of liver abscess. My friend and successor, Surgeon-General W. Burns Beatson, has just now written, in reply to my question, ' During the nine years I was civil surgeon at Chittagong, I never had a case of hepatic abscess.' Chittagong lies upon a volcanic line, much as Ischia does, having Seetacoond, a hot spring, close to its north, and a volcano, Barren Island, far to its south. At Chittagong, and for great distances on either side,

strong earthquake shocks are of frequent occurrence. I very narrowly escaped death in one at that place. Most of the West India Islands are of volcanic origin. Thus the two localities are as much alike as Monmouth and Macedon—and more so. I am, however, quite at a loss to account for the fact that there is no reason to believe that an indigenous case of liver abscess has ever occurred in the large district of Chittagong, and in its three hospitals, among the multitudes of malaria-sodden and dysenteric sick who are treated there.

Diligent search is still needful to show whether there are evidences upon which to rest a supposition that hepatic abscess is caused by a microzyme, a microphyte, a microzoon, or what not; which, as would seem to be the case in the West Indies, is not always present, is liable to return and again to disappear.

To the young Indian medical officer who is well grounded in pathology, I would say, Study attentively, and bear constantly in mind, with a view to bringing the suggestions to the test of your own observation, the original remarks by Dr. George Harley upon the probable influence of parasites or germs in the causation of liver abscess, which will be found at page 116 of vol. i. of the 'Medical Press and Circular' for 1886. Inquire into the causes which lead to the development of hepatic abscess rather in one hot climate than in another, and rather in one district than in another traversed by the same parallel. I would, following the lead of Dr. Harley, urge him to look fully into the question, does hepatic abscess depend upon the lodgment in the diseased organ of some animal or vegetable parasite, as Actinomycosis? seeing that there prevails some at present inscrutable law which rigidly confines each of certain orders of microzoa and microphytes to certain wide but perfectly defined tracts, as Bilharzia hæmatobia is confined to Africa, and as, in India, mycetoma and the embryo of Dracunculus are debarred from passing the western boundary of the Gangetic Delta.

Symptoms and Diagnosis of Liver Abscess.

When I first went to India, no ignorance was displayed in the differentiation of these cases; but, even when the great obscurity of many of them is allowed, it is very remarkable how frequently liver abscess was misunderstood some fifty years ago. 'What,' said an eminent Calcutta surgeon, as recently as 1848, of a colleague who was consulted in his wife's fatal illness, 'is it possible that he does not see that the case is one of liver abscess!' A few years earlier, a very accomplished physician, whose memory I revere so much that I will not name him, published a long series of cases of hepatic abscess in nearly all of which the diagnosis was wrong.

Certainly these cases are often so obscure as to puzzle men of great experience. A medical subordinate in Calcutta had maintained

a character for most exemplary respectability until he became dangerously ill, when it was discovered that, upon going home from work, he was in the habit of drinking nearly a quart of spirit every night. He had pneumonia of the *right base*, and coughed up a great deal of pus. If this case had occurred in England, everyone would have said 'pneumonic abscess,' of course; but, in India, 'hepatic abscess,' which was supposed to exist here, would be a safe opinion in ninety-nine out of every hundred cases of pleuro-pneumonia of the right base. Mr. M.'s case proved to be one of pneumonic abscess. I never saw or heard of a precisely similar case in India.

There is a very remarkable case by Surgeon-Major W. Johnson,¹ in which a tubercular abscess in the base of the *left lung*, opening into the pleura, simulated abscess of the left lobe of the liver.

Shortly before I left Calcutta, I saw, with my friend Dr. Joseph Ewart, a respectable middle-aged woman who had what very closely resembled a hepatic abscess, a very tender tumour, about as large as a goose egg, where the epigastrium and right hypochondrium met. The question was—cancer of pylorus and pancreas or liver abscess? The presence of enlarged glands in the right groin set this doubt at rest. But for this, I must admit that I should have remained uncertain whether the case was not one of liver abscess.

A well-conditioned northern sailor was brought to my ward. No one in the building could understand his language, so that we knew nothing of the history of his case. He was found to have pleuro-pneumonia of the right base. In a few days, he suddenly coughed up an immense quantity of pus of a slightly greenish hue. There was no evident hepatic enlargement. He recovered quickly.

I was completely deceived in the case of a middle-aged European whom I had full opportunity of examining. He was sent to my ward from Barrackpore, very ill. I understood that the case was considered to be one of hepatic abscess, communicating with the lung, in an intemperate subject. The liver was considerably enlarged upwards and also below the ribs in front, large quantities of blood-tinged pus were expectorated, coarse râles and gurgling pervaded the right lung. There appeared to be clear evidence of hepatic abscess finding exit by the right lung, which, I believed, had become much diseased in consequence.²

¹ *Madras Quar. Med. Jour.*, No. 10.

² In these cases, where rapid recovery does not occur, the structure of the lung almost always suffers considerable disorganisation. Thus, as will be seen in the case of my friend Mr. A., p. 643, lung mischief remained and caused death after the liver abscess had so completely healed that its vestiges escaped notice. In Waring's 7th case, a 'large abscess, eight inches in diameter, existed in the right lobe of the liver; it communicated freely with the right lung, and its walls, in the substance of the lung, were strengthened by a distinct red layer of lymph. At the upper part of the cavity, the substance of the lung was quite soft and disorganised, and contained several vomicae.' In Case 82, the lower part of the right lung

On examination, *p.-m.*, the liver was much enlarged and in an early stage of kirrrosis, but free from abscess. The pus came from phthisical abscesses in the right lung. Here occurs a point which is important to young medical men in India, where the visit is always in the early morning. In cases of phthisis and bronchitis, our patients have generally freed their lungs as far as possible of muco-pus fully an hour before we examine them—consequently, fluid sounds are much less distinct than they are later in the day. What I condemned my diagnosis most for in this case was the fact that I had not been sufficiently careful in examining the *left* lung. It had worked efficiently to the last. Had I examined it more closely than I did, I doubt if I could have discovered its very slightly diseased condition; but there was some disease here, which I told my class I ought to have looked for.

In 1856, I met with a case which signally illustrated the difficulty which may attend the diagnosis of a central liver abscess. I spent a few days in Madras, when a Presidency surgeon asked me to attend with the ship's surgeon a Bengal civilian of very high rank who had come down for his health and was returning by our steamer. I was told that he had suffered from pain in the hepatic region, and appeared to have passed 'gunpowder' from the gall-bladder. I had before known Mr. D. by sight. Now, his face was so much changed and pinched, giving the idea of the gravest visceral disease, that, after a few inquiries, I said that, in Bengal, we always suspected liver abscess in such a case. One day, on the voyage up, Mr. D. appeared much better than usual, and sitting well wrapped up on deck, was interesting a group of us much by his remarks on English poetry, he himself being a poet well known in India. Suddenly there was a moderate breeze, his aspect changed, and he began to shiver. We got him down to his cabin immediately. Except perhaps in my own case, I never witnessed such a shattering rigor—a true convulsion—he could scarcely be held upon the narrow berth, the massive woodwork of which shivered under him. This only occurred once. We placed him, on landing, in his own

'adhered to the diaphragm, and was in part converted into a sac filled with purulent matter.' In Case 85, there was, in the lower part of the right lung, 'an abscess the size of an orange, which communicated through the diaphragm with an abscess in the right lobe of the liver about the same size.' In Case 122, 'the right lobe of the liver and the lower portion of the right lung were occupied by an immense abscess. The lower part of the lung was completely removed, ulcerated off horizontally; and, laterally, the pleura, adhering to the ribs, formed part of the sac.' In a case which Surgeon N. M. Reid, A.M.D., reported in July 1883 in the *Lancet*, p. 96, a large abscess, the size of an orange, at the base of the right lung, communicated, by an aperture as large as a threepenny-bit, with a similar but larger cavity at the back part of the liver. Both these organs were studded with numerous other smaller abscesses. Very many other cases of very extensive lung mischief, occurring under these circumstances, might be given.

house and made him over to the care of Dr. Webb, his own medical man. Dr. Webb's hands searched his very meagre abdomen almost as thoroughly as if its wasted parietes had not covered it. There was then no pain whatever, and no enlargement of the liver. Dr. Webb made us feel two or three old hard ulcers in the colon which he told us had been long there. After thoroughly investigating the case, Dr. Webb said,—‘But for what you tell me of the history, especially of the rigor, I should deny the existence of liver abscess here.’ The patient rapidly sank. A day or two before his death, he passed some white, curd-like matter by stool. This led me to expect that the contents of an abscess would be voided. It however appeared, *p.m.*, that this matter came from the ancient ulcers in the colon. At the autopsy, Dr. Webb turned out the liver, and looking at me said, ‘There now!’ It was rather hyperæmic, but appeared free from blemish. I drew a long breath, and then, thinking of the rigor, replied, ‘Cut into it.’ Its centre was occupied by a recent abscess as large as a foetal head.

When we see the case early or where it comes to us so late that we have had no opportunity of watching its commencing progress, too much caution and observation cannot be devoted to it before we commit ourselves to a positive diagnosis. *Pain* is a very uncertain and variable symptom. I saw very little of the right shoulder pain of which so much has been written. Waring, who gives an invaluable analysis of the symptoms in his collection of cases, which ought to be read by everyone who desires to study the subject, finds that this symptom existed in 48 cases, and that, in 24 cases, it was expressly stated that it was absent. He concludes thus, ‘We are entitled to say that pain in the right shoulder is most frequently present when the abscess exists in the right lobe, especially when it is situated in its upper portion, but its presence no more necessarily implies that the abscess is in this lobe, or is in this portion of the lobe, than we are entitled to conclude that this lobe is not the seat of the suppurative process, *because* pain in the shoulder is absent.’ In my own case, I had no shoulder pain, but I had a most annoying uneasiness which I have not seen mentioned elsewhere, probably because, unless the patient happens to be an anatomist, his greater sufferings may cause him to overlook it. For about two inches above the clavicle, my *right phrenic nerve* felt like a sensitive cord painfully stretched. Where there is *inflammation of the hepatic peritoneum*, as there was in my own case, the pain is so great as to render even the slightest movement of the trunk nearly impracticable. I could only obtain some abatement of agony by lying like a corpse immovably on my back. Since then, my right side has been perfectly free from uneasiness, save once or twice when I have had an acute momentary pain, when coughing violently, as if adhesions had given way; and that, whenever I have bronchitis, the painful effects of long coughing are

felt much more on the right than on the left side. *Pain in the hepatic region*, in cases where the peritoneum is not involved, is a frequent but inconstant symptom. Large central abscess and multiple abscesses do not of necessity cause it, either when the patient lies quiet or when the hepatic region is palpated. In a large majority of cases, pain appears among the early symptoms, probably it is then what I felt—a sense of dull heavy tension as if a cannon-ball occupied the centre of the viscus.

It may be taken as a general rule that pain is greatest where the abscess encroaches on a serous surface—pericardium, peritoneum, or pleura.

Mr. Rogers, of Madras, and Mr. R. H. A. Hunter, of H.M.'s 37th Regiment, were inclined to ascribe chronic pain in the right hepatic region of old Indians to the colon rather than to the liver.¹ This remark is of considerable but limited importance. A *Friction Sound* may now and then be recognised in peri-hepatitis. *Difficulty in drawing a full breath* is a very frequent symptom.

Decubitus.—Waring shows that, of 35 cases in which this point is noticed, 10 patients could not lie on the left side without increase of pain, and 4 others found this the easiest position; 3 could not lie on the right side, and 3 others lay on this side easiest; 3 could not lie on either side, but were obliged to rest with head and shoulders much raised; 2 others, on the contrary, could lie on either side, and 1 found the back the easiest position. Two could not lie on the back and left side, and another could not lie down at all from a sense of suffocation. In this case, a liver abscess 'communicated with another large abscess in the right lung.' 'There were 8 or 9 ounces of serum in the pericardium.' It is self-evident that decubitus must be ruled in each case by the condition of the parts. In my own case, I could not lie on my right side while inflammation of the hepatic peritoneum was active, and an intolerable sense of weight and dragging prevented me from turning to my left side. As I have said above, I only found comparative ease when lying on my back.

A case by Dr. Nolan (*vide* p. 644) shows that *evidence of pericarditis* may exist when hepatic abscess is making its way into the pericardium.

Tensive contraction with pain of the abdominal muscles, especially of the right rectus, very rarely occurred in my own experience. Waring mentions, in a full discussion of this point, that he only finds it noticed as being present in 5 of his 300 cases.

Jaundice and Clay-Coloured Stools are not to be looked for as characteristic of liver abscess, seeing that, as a general rule, there is, in these cases, quite enough healthy structure remaining to carry on the

¹ *London Medical Gazette* for 1847, vol. i. p. 10.

functions of the liver. In some cases, the skin is sallow or dusky and the eyes yellowish; but this may arise from blood disintegration. Jaundice was present in only two of Waring's cases. In another case, where there was 'an extremely yellow suffusion of the skin,' the stools were 'healthy or dark green, the urine copious and surcharged with bile.' In only one of these 300 cases, the stools latterly 'became perfectly white and totally deficient in bile.' The skin was 'sallow,' the urine natural. 'Jaundice,' wrote Morehead, 'is very seldom present in' [Indian] 'hepatitis.' It did not occur in any of my own Bengal cases. I did not see any case of liver abscess in which the stools appeared to be deficient in bile. *Blood* is frequently observed in considerable quantity, with or without pus, in the *stools* when an abscess communicates with any part of the alimentary canal.

In many recorded cases, the *Tongue* is described as being foul or coated. It is often clean, especially late in the disease. I did not find any characteristic appearance, unless it was the white, flabby dyspeptic tongue.

Urine.—Retention or suppression; dysuria or strangury occurred in eight of Waring's cases. As might be expected in patients suffering from dysentery and from abscess in a vital organ, the state of the urine has frequently been abnormal; but, except where it contains pus (*vide* p. 645), there does not appear to be any state of this excretion which can be regarded as at all characteristic of liver abscess.

Rigor is a most important symptom when the physician happens to see it, as I did in the case of Mr. D. (p. 632), or to endure it as I did myself. There is nothing else in nature like it—so terrible is its severity and so strong the convulsive agitation of the body. I, on one occasion, had an ague fit more severe than I have ever seen ague in another; but, here, the shivering was moderate as compared with the rigor of hepatic abscess. I doubt its occurrence in cases of multiple ichoræmic abscess. We see it in other cases where tensile force is suddenly applied, as in passing the catheter. Waring finds that rigor was mentioned as present in 25 cases and absent in 22.

Vomiting or *Nausea* was present in 74 of Waring's cases, and absent in 10. One day, when the peritoneal pain kept me as immovably on my back as if I had been lying in my coffin, the horrible idea occurred to me, 'I am going to vomit.' I do not think that I was ever so panic-stricken—I dreaded the exertion so vividly. As I began to retch, the suffering was great. Then came the consolatory feeling, 'I am doing it much better than I expected.' Afterwards, the pain was somewhat abated. I think that it is Mr. C. Macnamara who says that, when vomiting occurs, the strong surrounding muscular pressure squeezes the blood out of a congested liver as if it were a sponge. Flatulence and eructation are frequent symptoms.

Risus Sardonicus.—I saw this once in a patient of Dr. Thomas Addison's in whom a fold of diaphragm, drawn up by a contracted lung in pleural effusion, was wounded by an exploring needle. I was, therefore, able to recognise its very marked presence, throughout a whole day, in one of my European patients in whom hepatic abscess progressing upwards had evidently caused an unusual amount of diaphragmitis. Much to my regret, the dying man's friends insisted upon taking him home that evening. *Hiccup* is frequent in liver abscess.

Enlargement of the Liver.—In every suspicious case, most careful palpation and percussion, over and beyond the natural area of the liver, will, of course, be made early in the disease. I now and then found enlargement upwards—scarcely ever downwards in the hypochondrium or epigastrium—of course not taking into account cases where abscess-pointing causes a dense swelling thereabouts, which is not usually liver. Waring finds that there was evident enlargement of the liver during life in 90 cases, whilst, in 11 others, this did not exist. Late in the disease, when the accumulation of pus is very large, the following conditions, as cited by Waring, are frequently observed:—

‘In Case 11, the right side bulged out, and was conspicuously enlarged compared with the left side, which appeared quite flat; in Case 47, the intercostal spaces were much increased in breadth; in No. 74, the right side was elevated, and the nipple was on a lower level than that of the left side; in No. 79, there was a bulging of the ribs on the right side, and a want of depression in the intercostal spaces; and, in No. 122, there was some fulness, with blue veins meandering over the right hypochondrium.’ In making this examination, we, of course, notice how far the expansive movement of the right side is affected in respiration, and ascertain whether there is any spinal distortion. In two of Waring's cases, 13 and 95, a liver abscess had carried the diaphragm up to the level of the 4th rib. In 82 and 94, it reached the 3rd rib; and in 79, it reached the level of the 2nd rib.

A very little experience gives us far more power of judging these cases by *the Aspect of the Countenance* than can be communicated in any written description. A glance at one of these pinched anxious visages affords us a lasting memory which can always be applied again. The excellent apothecary of a London hospital, whose education had not comprehended auscultation, used to shake his head and say: ‘He is VERY ill—I think it is lung!’ So, when, in India, we see one of these shrunk, wizened countenances, we say: ‘He is very ill—it is probably liver,’ and then we proceed to find out whether it is, in reality, liver, which it occasionally is not. This aspect is always an expression of very grave visceral disease. In 30 of Waring's cases there was noticed ‘countenance expressive of anxiety, anxious, shrunk or collapsed; pale, livid, or parchment-like.’

When the pus of a liver abscess is voided through the lung, it may

sometimes be found to contain *débris of liver tissue* (Fenwick). I saw *bile in the sputa* only in the cases of Mr. A. and of the Northern sailor (pp. 643 and 681); its appearance has been rather frequently noticed by others.

Temperature.—I believe that, when we take full care in excluding other causes of increased evening temperature, the clinical thermometer affords us an almost infallible guide in the diagnosis of liver abscess. It has always been noticed that, in an advanced stage of this disease, the patient's condition is 'Hectic.' Thus, in Waring's cases, there was hectic fever in 34; cold sweats, or profuse or clammy perspirations in 72; evening or occasional exacerbations of fever in 22; prostration, exhaustion, or great debility in 74. In all my cases, latterly, a temperature chart formed part of the record, and, in all, the evening temperature was abnormally high, the rise being generally very moderate but distinct and steady until the case took a validly favourable turn.

Profuse night sweating occurred late in my own illness. One morning, towards daybreak, I felt absolutely convinced that I was dying. The sense of debility was extreme, and I was almost unable to swallow food. My medical friends advised me to go to sea, but I said that I could not muster strength enough. In a day or two, however, I was removed, in the cool of the evening, to Spence's Hotel. I had a delightful sleep throughout the night, and awoke with a good appetite. I recovered quickly without a bad symptom; night-sweats are, however, generally among the signs of approaching dissolution in cases of liver abscess.

When going over the above enumeration of symptoms it will probably be said, 'You have nearly succeeded in showing that all the leading signs of liver abscess, with the exception of the temperature, are most capricious and uncertain, and you have given us no distinct clue to the puzzle of differentiating this disease.'

Undeniably, many of these cases are very perplexing, even to the most experienced. Still he who gives sufficient patience and thought to his cases will seldom be mistaken. We usually perceive the nature of the malady clearly where we can observe the symptoms from the beginning of the illness, and can watch each symptom as it occurs, as I had the questionable advantage of doing in my own case. Save that we ought—upon the appearance of the very first symptom, say of weight in the hepatic region, which should be inquired for daily in every case of dysentery, especially for some time after the flux has ceased—to prevent the occurrence of further symptoms by prompt treatment. When, at a more advanced stage of the disease, the patient is intelligent enough to give, in reply to our questioning, a clear account of his early symptoms; that and his present condition will often guide us safely. When the case comes to us late, there ought to remain

little doubt. We find him hectic, wasted, 'very ill,' with a raised evening temperature. We find that all other causes of hectic, phthisis, splenic abscess, extensive suppurative disease elsewhere, &c., &c., are absent. We learn that two or more of the symptoms analysed above, hepatic pain, bulging, shoulder-pain, rigor, &c., &c., are or have been present. '*Such symptoms,*' says Dr. John Macpherson, '*are more valuable taken collectively than individually.*' I doubt if any three of the principal symptoms characterising liver abscess, in addition to hectic, can be found combined in any other disease.

Morbid Anatomy of Liver Abscess.

It does not happen to everyone, in his Indian dead-house experience, to have opportunities of tracing the process of formation of liver abscess from its initial stage. A few pathologists have done so ; but, if each observer who meets with a transitional specimen of idiopathic Indian abscess would now publish a description of its microscopical and other characters, he would do a useful work.

Morehead has given¹ a good account of the morbid conditions of liver tissue which end in the formation of abscess. Dr. F. N. Macnamara has made some very careful observations upon the structural changes which precede liver abscess and upon those which immediately lead to pus formation. He insists upon 'a granular degeneration of the cells, attended, in the more advanced stage, with a deposition of intercellular granular matter.' This change may be present in livers in which abscesses have not formed, but, as a general rule, subject to exceptions, livers in which dysenteric abscesses are found present a granular change. He considers that 'such a precipitate of albuminous molecular matter, both inter and intracellular, may arise from repeated attacks of slight congestion of the liver, or from long-continued engorgement of the organ.' The colour of the liver is very constant and characteristic, an uniform pale-drab colour. Such a liver may be found much congested ; but, upon the escape of the fluid, the drab-colour appears. Very early, we find, scattered through the substance of the organ, one or more degenerating patches varying in size from that of a pin's head to that of a large pea. These are paler and softer than the surrounding parenchyma. These present the first discernible change which precedes the abscess. In the next stage, these patches may be as large as a walnut ; but, if we do not hit the central portion, we see but a soft, straw-coloured spot fading off into the neighbouring substance, no congestion, no halo of redness. The centre of the area of degeneration is liquescent or completely fluid, and puriform in appearance, but the fluid may not present a single pus

¹ Pages 327-9.

corpuscle. There may be a central cavity, the fluid contents having disappeared; but, whether the cavity does or does not contain fluid, its parietes are soft, ragged, and spongiform. In the third stage, there is a surrounding halo of congestion, which, he considers, does not precede the local degeneration. From this condition, (1) the disintegrative process may spread, more and more tissue breaking down, and the neighbouring cavities uniting to form an immense abscess; or (2) the degenerated patch or cavity may become encysted by lymph thrown out by the vessels of the halo, the fluid may be absorbed, and the dead tissue dry up into a semi-solid mass.¹

Terminations of Liver Abscess.

In this disease, Nature displays the fullest ingenuity of her curative art. Although, early in any given case, the prognosis is very unfavourable, hope always attends liver abscess. The pus may find its way to discharge by various routes, and many of the most fortunate cases are those which the physician allows to take their own course with-resorting to his *nimis diligentia medendi*. I long considered that a thoracic aneurism or a hepatic abscess makes its way lawlessly as a bullet or a sword-blade does. But this is altogether an error. True, in both, there are various modes of advance, but full experience shows that the variety of these is finite, and that the occurrence of each of these numerous (thirteen or more) modes is ruled by definite laws and determined by various ascertained conditions, of which situation and anatomical surroundings are the chief.

(1) *Liver Abscess may become encysted, its contents undergoing more or less complete absorption.* I ought to know more about this termination than I do, seeing that it probably occurred in my own case. Having, in an illness lasting six weeks, gone through what all the medical friends who saw me and I considered to be every characteristic symptom of liver abscess, except its discharge, I almost suddenly recovered, and, save in one or two points, with which the abscess has had nothing to do, have remained without any sensation telling me that I have a liver during the thirty years which have since elapsed.

The preparations numbered 532-33-34, in the Pathological Collection of the Calcutta Medical College, illustrate this condition. They display cysts imbedded in the liver substance, which has evidently contracted around them. They all enclose calcareous deposit, which, in two, appears to have been of mortary consistence, and in the third is hard and as large as a walnut. Cases like my own are not very

¹ I. A., No. xv. p. 1. For interesting details of the microscopical appearances in a case of multiple Liver 'abscess' in dysentery, by Dr. Alban Doran, *vide* Sir Joseph Fayrer's *Lettsonian Lectures*, p. 92.

common. I knew a remarkably hale-looking middle-aged European gentleman whom my friend Dr. T. W. Wilson watched through a similar recovery. I attended a lad of European parentage, studying at Bishop's College, who had every symptom of liver abscess, save that no pus made its appearance, although it was most carefully looked for. I heard of him, for many years subsequently, as an active missionary in the Upper Provinces.

Kenneth Mackinnon gives the case of a man, in the 1st Bengal Fusiliers, who, ten years previously to his death, had an acute attack of hepatitis. The presence of matter was diagnosed, and it was believed that he would die. A round tumour was discovered protruding from the surface of the liver. 'It was found to contain pure pus in a thick cyst or sac. The cyst was like leather.'

The presence of these cysts is not always free from danger. In the liver of an European who died at the Medical College Hospital of dysentery, Dr. Macnamara found, in the right lobe near its upper edge, a congested spot about the size of a walnut. In the centre of this, there were two white masses, each the size of a large pea, separated from one another by a little fibrous tissue. They had the appearance and consistence of cream cheese, and each was enclosed in a fine membranous cyst. There was a little moisture between the outer surface of each cyst and the parenchyma. Under the microscope, the contents of the cysts proved to consist chiefly of fat, mixed with granular matter, and the débris of liver cells. Dr. Macnamara adds: 'These cysts, with their contents, presented the happiest termination of liver abscess, but they appeared to have become the focus of a new congestion under the disturbing influence of the attack of dysentery of which the patient died.'

C. B., when at about the age of 35 years, suffered from every symptom of central liver abscess, save that no pus appeared. He subsequently gained a very high position. I spent a day with him, in his retirement in Gloucestershire, twelve or thirteen years before his death. He was then a great walker. He died at about the age of 62, it was said of 'lung disease.' I do not believe that he was a phthisical subject, and I am almost sure that the encysted abscess was to blame, as it certainly was in the following case.

A Bengal civilian retired to England, having, I believe, served his full time in India. His friends at the club noticed that his appearance was always very sickly. After about six years, he was advised to try hydropathy—pleuro-pneumonia of the right base occurred, and he died from the effects of the acute attack. The autopsy revealed a large encysted liver abscess, of such age that its walls were cretaceous. Some irritation had caused the mischief to spread upwards and to involve peritoneum, diaphragm, pleura, and lung. It appeared that, as often occurs in acute cases of liver abscess, the diseased mass was

obeying a *nisus* to make its way upwards, seeking discharge through the lung.

Cases in which liver abscess appears to be going in quietly for isolation and cure are apt to do very badly when the sufferers presume too early that their recovery is complete.

My friend Dr. W., to whose admirable research in the field of Indian pathology this collection of facts is very largely indebted, became the subject of liver abscess while in almost constant attendance at a daughter's sick bed. I had no opportunity of seeing him until I went down to bid him farewell upon the deck of a P. and O. steamer. Apart from my concern for the suffering of a dear companion, I was literally 'taken aback' by the change which had occurred in his appearance. His naturally full countenance was so shrunk and sharpened as to remind one of a fox. I never saw a face which displayed so characteristically the pinched lividity which betokens grave visceral disease. Still I was afterwards told that he recovered strength so greatly, on the six weeks' voyage, that, although no longer a young man, he used to climb the rigging and sit in the tops. He, however, went to a pleasant place in England and there speedily died.

My colleague Dr. A., one of India's greatest botanists, was sent home for hepatic abscess. It was said that he considered himself so completely recovered that he went in for a too athletic life,¹ and lived for a very short time.

It is generally considered that so-called 'pyæmic,' multiple abscesses of the liver are inevitably deadly. They probably are so in a large majority of cases, but I met with an instance in which they had healed. A European sailor in my ward suffered from acute dysentery. His stomach was absolutely intolerant of ipecacuanha, (Morehead observed that, when this drug cannot be borne in dysentery there is usually liver disease), and he died and I found the cicatrised remains of numerous abscesses scattered nearly throughout the liver. Unfortunately, we did not preserve the whole organ, consequently my friend Dr. Ewart's description of the preparation, No. 516—'remains of two old small centrically situated hepatic abscesses. There is a considerable amount of fibrous deposit around both. There were no recent abscesses'—does not set forth the fact that these abscess cicatrices were very multiple.

In Mr. Sibthorpe's Madras Catalogue, Prep. 79, Series 8, is 'section of liver presenting several cicatrices of abscesses. Subject a European sergeant who had been under treatment six years previously for a severe attack of hepatitis with well-marked symptoms of abscess.'

Judging by the harmlessness, thus far, of my own cyst, it appears

¹ I cannot quite separate in memory the history of his case from that of the very closely similar one of another colleague of my own—Dr. F.; but I believe that Dr. A. was considered to have brought on a fatal relapse by rowing in sea water.

to me that the operation of hepatotomy could only be needful if such a chronic cyst should make its way downwards and point in the right hypochondrium, an occurrence which I have neither seen nor heard of.

(2) Among the most usual positions in which a liver abscess points are—*anteriorly, in the right hypochondrium, at the epigastrium, or even as low as the umbilicus*, but, in the last case, the pus has doubtless burrowed. As in the following case, there is sometimes more pus in the abdominal wall than in the original abscess. An elderly native man came into my ward with a fluctuating swelling, as large as two fists, extending from the xiphoid cartilage nearly to the umbilicus. He died before anything could be done for him. We found that the outer swelling was caused by a large collection of pus between the abdominal muscles and the integument, and there was a slightly cupped suppurating surface the outer edges of which adhered to the abdominal walls within. This was a liver abscess which had discharged itself through an ulcerated opening in the linea alba into the anterior cavity. Thus in a vertical section, the two cavities with the communication would have presented an hour-glass outline. It was beautiful to see how much in the way of repair nature had effected in this case. Had the patient been stronger, the outward abscess might have been opened and then the liver abscess would have become apposed and completely adherent to the abdominal wall within. I have seen in other cases this tendency of liver abscesses which advance to a wide surface, say diaphragm or abdominal wall, and then discharge themselves, to lose the wall which was foremost and to become mere cups and then plane surfaces, so that we must not always look for traces of a sac in seeking for the vestiges of an abscess cured by evacuation. Nothing may remain save more or less adhesion of apposed surfaces.

We must not suppose that, because the bulging of a liver abscess appears in the epigastrium or hypochondrium, it is safe to open spontaneously or to be opened there. In the case of a poor Afghan lad, sinking from dysentery, there was a defined fluctuating swelling as large as half an orange in epigastrio. I had not the slightest doubt that the pus was seeking an outlet at that spot, but it was clear that, as yet, there was no adhesion between the sac and the abdominal wall; besides, the case was evidently hopeless. In a day or two, the hemispherical swelling entirely disappeared; and, a little later, at the *p.-m.*, I found that the abscess was at least three inches to the right and much above the spot at which it had presented itself. It appeared probable that, as the poor fellow's fluids were drained away, the mass of his liver shrunk and thus the non-adherent sac altered its position.

(3) Many liver abscesses point *between the ribs, at their greatest bulge*. Here the bone is very apt to suffer and the integument to slough, especially when, as is frequently the case, there has been time for the

pus to burrow extensively before we see the patient. Pus which finds an exit between the ribs may burrow to an almost unlimited extent. Waring cites a case by Portal¹ in which an abscess of this kind opened into the axilla.

(4) Abscesses situated at the highest convexity of the liver and in its centre have a great tendency to find exit *through the diaphragm into the right pleural cavity*. This most serious accident occurred in 14 of Waring's 300 cases; or *through the diaphragm and base of the right lung*. In these cases, when the collection of pus is large and the opening is into a considerable bronchial tube, the first gush may suffocate the patient, but Waring has collected ten of these cases which ended in recovery, and I have watched several to a successful termination. When an expedition to China was on foot at Calcutta, a medical officer, much occupied in making arrangements for shipping his stores, came to my office on a very hot day and got over a good deal of business. As he rose to leave, he said, 'To add to my troubles, I coughed up the contents of a small liver abscess yesterday.' To my astonished look, he replied, 'Yes, Dr. Edward Goodeve thinks so.' He looked out of condition; but the case appeared so extraordinary that I inquired of Dr. Goodeve (whose mastery in the diagnosis of Indian disease has never been surpassed in my experience). He replied, 'Yes, he considered that the pus which Mr. D. showed him was from a liver abscess.' Mr. D. went to China; and told me, in after years, when he was a remarkably healthy-looking man, that his liver had not given him any further trouble.

Dr. John Macpherson² has 'known an iron-worker burst an abscess' [liver] 'through his lungs when at work, the presence of which was not suspected.'

A sepoy recruit, whose case is cited by Waring, was brought to hospital collapsed, having frequent calls to stool, and complaining of pain in the right side. He died $5\frac{1}{4}$ hours after admission. An abscess containing 34 ounces of purulent matter lay between liver and diaphragm. This authority also cites the fatal case of a European soldier, in whom an abscess had burst into lung *when he was at his duty*.³

In some of these cases, there is speedy recovery (as happened in the case of a northern sailor in my ward, who was very nearly suffocated while coughing up about three pints of pus in a few minutes; see p. 631). In others, recovery is very tedious. In some, there is long illness followed by death. Mr. A., a fellow-student of my own, expectorated the contents of a large hepatic abscess. I saw him, with Dr. C. Palmer, for a considerable time. When he became strong enough, some expectoration still continuing, we placed him on board a sailing

¹ *Anat. Méd.*, t. v. p. 304.

² Quain's *Dictionary*, Art. 'Hepatic Abscess,' p. 830.

³ *Op. cit.*, p. 143.

vessel, and he was taken to England round the Cape. At home, his condition improved, for a time, so much that, as he wrote to me, he was just able to take a gun and drag himself along in search for game. He said, 'Whenever I drink water, which I do not like, I spit nothing but pus; if I drink wine, which I do like, although it be only a teaspoonful of claret, I spit nothing but blood.' When he died, those who made the autopsy said, 'The Indian doctors mistook pneumonic abscess of the right base for liver abscess.' I, however, had seen all the characteristic evidences of hepatic abscess, one of which was that the sputa which I examined contained much bile. The liver abscess must have healed completely (although I am as certain as if I had seen it, that there was adhesion between the upper part of the liver and the diaphragm), but the lung mischief remained and advanced.

(5) A most disastrous complication, of rather frequent occurrence in these cases, is where *liver abscess opens into the pericardium*. See Morehead, p. 345. References to six of these cases are given by Waring.¹ See also a seventh case,² and an eighth by Dr. H. V. Carter.³

Dr. Curran notices a ninth and a tenth case (in a woman and in a staff officer), and cites other cases.⁴

This accident is especially liable to occur where the abscess occupies the *left* lobe. Where the opening is at first minute, death may not be sudden. In a case reported by Dr. Bentley, it was considered that the pericardium was opened thirteen hours previous to death. Dr. William Nolan has recorded⁵ a rather similar case. A Scotsman, mate of a steamer, *æt.* 25, was admitted to the European General Hospital, Bombay, with signs of pericarditis. 'The area of præcordial dulness was increased, and the friction sounds of pericarditis, with tumultuous action of the heart, were easily recognised. His countenance was expressive of anxiety, and respiration was almost impossible, except in an upright position. The liver did not present any evidences of enlargement, and his case was set down for one of pericarditis. On the morning of the third day, it was evident that effusion into the pericardium had taken place, to a considerable extent, during the night. At about two o'clock, he was seized with severe pain over the apex of the heart, while the dyspnoea became more oppressive. In this condition, he lingered about four hours, and died in the evening, evidently suffering great pain. The pericardium contained twenty ounces of a sero-sanguinolent fluid. The heart was covered with recent lymph. There was adhesion between the surfaces posteriorly and at the apex, the fluid being confined to the anterior and upper parts of the sac,

Op. cit., p. 133.

² *Bombay Med. and Phys. Trans.* for 1853, p. 305.

³ *Ibid.* No. 8, N. S.

⁴ My contribution towards a clinical study of Hepatitis in India, *I. M. G.*, vol. xv. Nos. 9 to 12, and vol. xvi. Nos. 2 to 4.

⁵ *I. M. G.*, vol. ii. p. 177.

which was increased to nearly three times its normal size, and was very vascular throughout. Near the apex of the heart, at the attachment of the pericardium and diaphragm, was an opening with thin ulcerated edges, easily admitting the little finger. Through this, pus was seen making its way from a circular abscess, large enough to contain a cocoa-nut, in the left lobe of the liver. Usually, death is instantaneous. This fatal accident occurred to a native man in one of my wards. He was seen to leave his bed, and to begin to cross the ward; midway, he gave what seemed to be a caper, turning half round and falling dead.

Dr. Joseph Ewart¹ thus describes a preparation in the Calcutta M. C. Museum: 'Liver, heart and pericardium, and large intestine of a patient, exhibiting a hepatic abscess of the left lobe opening into the pericardium, and dysenteric ulceration of the colon. The patient was a European, 21 years of age, admitted into hospital April 12, 1866, and died 26th idem. This man died suddenly and unexpectedly, from the influx of unhealthy pus into the pericardium.'

Mr. William Dymock² narrates the case of a German seaman, *æt.* 25, in the European Hospital, Bombay, who had partially coughed up the contents of a hepatic abscess through the right lung. 'After a fit of coughing, sudden collapse occurred.' An abscess of the right lobe of the liver had, as so often happens when hepatic abscesses are in process of recovery, become 'of a saucer-like shape, and formed part of a common suppurating sac occupying the lower and middle lobes of the right lung.' The pericardium was much distended with pus, which had entered through 'a stellate rupture.'

(6) Dr. J. Mouat, senior, and other early observers³ stated the, at first sight, perplexing fact that they had seen *abscess of the liver terminate by a discharge of pus by the urinary bladder*. Here absorption, which some fancied might have occurred, was undoubtedly out of the question. The state of the right kidney was, of course, considered. Webb noticed that one of the many lobes of the liver of an orang-outang was disorganised by suppuration, which implicated the right kidney in the same abscess. The heart was 'inflamed and stuffed with fibrinous coagula.'⁴ Specimen 531 in the Calcutta Museum shows adhesion of the liver to the diaphragm and kidney of the right side. An abscess made its way through the cellular tissue, and, Dr. Webb thought, through the structure of the kidney into the lumbar region.⁵

In Waring's case, No. 32, 'the sac of the abscess was found adherent to and communicating with the right kidney.' In Case 253,

¹ *I. A.*, No. xxi. p. 308.

² *I. M. G.*, vol. i. p. 32.

³ Mouat, *C. M. T.*, vol. iii. part 2, and *Madras Quar. Med. Jour.*, vol. ii. p. 20, and Conwell, *Diseases of the Liver*

⁴ *Path. Ind.*, p. 264*

⁵ *Ewart's Catalogue*, p. 123.

'an extensive abscess of the liver, extending downwards, completely destroyed the right kidney.' Waring notices the absence of information with regard to the state of the urine.

In all probability, the appearance of pus in the urine is, in most cases, not attributable to the coincident liver abscess at all, but to the presence also of an ichoræmic renal abscess. Such renal abscesses have been noticed by Dr. Chuckerbutty, who wrote :¹ 'It is interesting to notice, in Case 18, that abscesses in the liver which, during life, had been known to empty themselves through the diaphragm and lung, had become completely cicatrised; that evidences of inflammatory action in the liver were still present in its numerous adhesions, and the induration and flattening out of the substance of the left lobe. The *pelvis and substance of the right kidney were the seat of an existing abscess*, a complication of dysentery I had observed before.' Pus does not appear to have been noticed in the urine, but it is not improbable that it would have been found if looked for.

In 6 out of 45 of Waring's cases, in which the condition of the urine was observed, it was 'purulent, containing purulent matter, or a puriform deposit.' He remarks that, 'in those cases in which the urine was purulent, there was no communication between the abscess and the kidneys.' On referring to the details of these six cases, however, I find that the condition of the kidneys is only mentioned in one, as being 'diminished in size.' Dr. Waring has entered into a statement of facts (p. 196), by which he is led to the conclusion that 'the contents of the abscess were absorbed by the hepatic vessels, were thus carried into the circulation, and subsequently eliminated by the blood-vessels.' This important pathological question must still be held *sub judice*; but I think that, at present, the preponderance of facts is in favour of the opinion that the pus found in the urine usually comes from ichoræmic renal abscess. There is great need for further data, showing (1) whether the puriform urine actually contains true pus or merely phosphates [Budd], and (2) what is the condition of the kidneys in these cases.

Dr. W. Rutherford has given² details of a very remarkable case in which an extensive *abscess of the right kidney simulated abscess of the liver*. The *left* kidney was somewhat larger than natural. Several very circumscribed abscesses were found in the cortical structure. The *right* kidney was in some measure buried in the mass of adhesions posteriorly, mixed up with purulent matter. The cortex contained a number of small abscesses, one of which, larger than the others, about the size of a hen's egg, communicated with the purulent collection in the surrounding cellular tissue. The kidney was nearly twice its

¹ 'Cases Illustrative of the Pathology of Dysentery,' *I. A.*, No. xix. p. 137.

² Cited in 'Annals of Naval and Military Surgery,' from *Army Medical Report* for 1861.

natural size. The cortical and tubular portions were scarcely distinguishable from each other. None of the abscesses communicated with the calices or pelvis. The largest abscess communicated with another external to the organ; thence it had found its way, along the crura of the diaphragm, posteriorly to the liver, into the right pleura and, ultimately, into the bronchial tubes. It also appeared externally, but the extract which I cite does not show where. The perforation in the diaphragm occurred in the muscular portion, at about the centre of its right lateral half, was oval in shape and about an inch long.

(7) *Evacuation of liver abscess through a bile duct* has been repeatedly observed.

(8) *Communication with the gall bladder* had occurred in Waring's Case 160.

It is frequently noticed that large vessels, generally portal, traverse liver abscesses. A native woman was sitting up in bed in my ward; she sank back and died immediately. It was found that a large blood-vessel had given way on the wall of an enormous central liver abscess. The preparation is numbered 514 in Ewart's Catalogue. *Vide* also Waring's 222nd case, a large branch of the hepatic vein traversed the upper part of an abscess, 'part of its coats was removed by ulceration;' also his cases 47, 226, and 258. In Morehead's 137th case, 'Thin pus was seen to flow freely from a large branch of the hepatic vein which could be traced to the abscess, with which it communicated.'

(9) *Escape of pus into the peritoneal cavity* is a not unfrequent accident. Here death is not usually so rapid as it is in cases of escape of bile and fæces. Waring gives (p. 134) a valuable analysis of cases terminating in this manner. In a few exceptional cases (as in one of Twining's where the patient survived twenty-six days), life is singularly prolonged, but Waring shows that 'the average duration of life, after the abscess has burst into the abdominal cavity, is about three days.'

(10) *Communication with the stomach* is not very rare.

(11 & 12) The abscess may communicate (rarely) with the *duodenum*. In Waring's Case 241, a liver abscess 'had opened into the duodenum just below the pylorus.' A similar case is mentioned in Dr. Leith's 'Deaths in Bombay,' as occurring in 1855; or, not infrequently, into the *colon*, the contents being voided by stool. This appears to have occurred in eight out of twenty-five cases of spontaneous recovery collected by Waring.

(13) In one of Allan Webb's cases (p. 278*) a liver 'abscess communicated with the hepatic vein at its junction with the vena cava.' *Vide* a questionable case, cited by Waring from Conwell, No. 223.

It is noticeable that, in these thirteen terminations, nature's arrangement, however imperfect it may be in many cases, is essentially curative in the majority: by isolating and partially absorbing the abscess,

or by emptying it into the stomach, bowel, or bile apparatus, or through the lung or thoracic or abdominal parietes. In only two rather frequent terminations—into the pericardium or peritoneal cavity—is it inevitably fatal.

As may be expected where more than one considerable abscess exists, the pus is liable to seek exit by *more than one course*. In his early career in India (Bombay) a relative of my own had an abscess or abscesses which found vent through the lung and in the hypochondrium. Waring has collected cases in which the discharge was by colon and hepatic ducts; stomach, duodenum and hypochondrium (pointing); lungs, and abdominal cavity.

Prophylaxis of Liver Abscess.

Strict temperance and regularity in living do not absolutely secure exemption from liver abscess and other hepatic diseases in India; but, undoubtedly, he who is moderate in all things is safest. Dr. Waring shows, in his admirable statistics of Indian disease,¹ that, in six European regiments serving in Madras, the percentages to strength of admissions for hepatitis were, teetotallers, 5·777; temperate, 5·766; intemperate, 10·191.² Nearly everyone must have heard Dr. Gregory's story of the officers, long subject to liver complaints, who, having been imprisoned by Hyder Ali upon a diet allowance of a pice (halfpenny) a day, returned in good health and found that their messmates had all died of good living. Noticing this, Mr. J. Murray cited³ a case which had come under his own observation. An officer had a severe attack of hepatitis which compelled him to go to England. After a two years' residence at home, without any improvement, he sailed again for India, and, during the passage, was wrecked on a desert island, where he remained for several months, living almost exclusively on penguin eggs. Under this regimen, in spite of exposure to the weather and the absence of every comfort, his liver complaint left him, and had not returned.

A very interesting observation is made in the 'Madras Medical Topography'⁴ regarding health at Pondicherry. It is noticed, as a fact of great importance, that hepatic complaints, especially of an acute form, are entirely unknown amongst the French inhabitants of this place. This exemption from a disease so prevalent and fatal amongst the British residents in India, is attributed to the French mode of living being so different both in regard to food and drink; the English consuming much animal food, malt and spirituous liquors, and strong wines; whereas the French live more on vegetable food and curries, and use little else than the light wines of France. So Chisholm

¹ *I. A.*, No. vi. p. 484.

² See *Influence of Habits of Life in the Causation of Liver Abscess*, p. 617.

³ *Bombay Med. and Phys. Trans.*, vol. i.

⁴ Centre Division, p. 66.

wrote that, when the worst form of yellow fever raged at Grenada in 1791, the utility of prudent living 'was remarkably illustrated by the almost total exemption of the French inhabitants from the disease. Their mode of living is temperate and regular in an uncommon degree. Animal food and strong liquors are very moderately used by them; and vegetables and small considerably acid wine chiefly compose their diet. Their passions are seldom excited to any degree bordering on excess; and their minds seem, in general, tranquil and actuated by a vivacity which nothing depresses, and which, indeed, is peculiar to themselves.' He adds: 'The same exemption, doubtless from the same causes, was observed at St. Pierre's, Martinico, when the pestilential fever was imported into that city from Philadelphia, towards the end of 1793.'

Medical Treatment of Liver Abscess.

In every case of liver abscess, the necessity for *removing every trace of bowel disease, and for detecting and dealing with any taint of scorbutus and malarial cachexia is of paramount importance.*

Beyond this, when the abscess has formed, we can do little, medically, for the patient beyond attending to his general hygiene and 'supporting' him.

While he should, of course, be carefully guarded against chill, his room should be as cool and airy as possible. I have seen death, by exhaustion, determined by the occurrence of a great rise in the temperature. Quinine must be given as a nervine tonic and as a corrective of that malarious taint which is very rarely absent in Indian disease.

Hydrocarbons must be abstained from as much as possible. No extra work of any kind must be put upon the already more than disordered function of the liver. In these cases, when the drain upon the system is evidently wearing the patient out, the temptation to give wine is strong, but must be resisted. The remark of my friend Mr. A. that the very smallest quantity of wine converted the expectorated matter from pus into blood is very significant. In Waring's case No. 47, there was 'excruciating pain in the stomach, especially after taking wine or beer.' None of my patients with hepatic abscess had wine. Although they were made a great many years ago,¹ I consider that the following remarks, by Dr. John Murray, are worthy of all attention. Speaking of the treatment of hepatitis, he says: 'It is incalculable the mischief that is done by indulging the sick and convalescents in extra food, wine and beer. I am very careful in my dieting, and place nearly as much confidence in that as in medicine. . . I put all patients with acute disease on spoon diet on admission. . . In very severe cases, merely a little tea, or lemonade, or thin sago, or arrow-root, is allowed for the first two or three days. When acute inflammatory

¹ *Madras Quar. Med. Jour.*, vol. i. p. 167.

symptoms have been completely subdued, milk diet is allowed, and generally continued till the cure is radically effected; when low, a chicken diet is given, with a measure of wine' [to which I object, N. C.]. 'In chronic cases, I consider that the cure almost entirely depends on keeping the patients for a long time on milk diet; and I have often witnessed the bad effects of any deviation from it—such as protracted convalescence, chronic persistence of the morbid actions, and relapse.'

Anything more absolutely unwise and cruel than the expedient of sending away a patient with liver abscess to die miserably on ship-board can hardly be imagined. I have, on two occasions, watched the sufferings at sea of these unfortunates. I know of instances in which persons with liver abscesses have appeared to improve on the voyage; they would probably have done still better in their own quarters. A sea voyage may assist recovery in hopeful cases of dysentery, but, in liver abscess, ship-life cannot, in my belief, prove beneficial.

Surgical Treatment of Liver Abscess.

It has long been the practice of Indian surgeons to endeavour to effect the discharge outwards of liver abscesses. We are told by H. H. Goodeve¹ that, in 1772, out of ten cases in which hepatic abscesses were opened, Curtis saved two; and, in 1773, four of his patients recovered out of fourteen. We read also of many successful cases of a similar nature in other writers of the same date. The following quaint entry appeared in the 'London Chronicle' of March 9, 1786: 'Sir John Burgoyne is known to have suffered much in health from his late affair, and some time ago he underwent the severe operation, common in India, of having his liver scraped.' It is added that 'the intelligence of Sir John's death came to England by the conveyance of the black merchants.'² When I first read this paragraph, some years ago, I thought that this was probably the popular idea of the treatment of liver abscess by incision; but, of late, I have heard of operations upon the liver which almost come up to the old newspaper reporter's idea.

Annesley recommended an incision from two and a half to three inches long, care being taken that the opening should not extend beyond the limits of the adhesion. The pus having been evacuated he filled the cavity with lint.³

As I have before said, nature often cures liver abscess more effec-

¹ *Sketch of the Progress of European Medicine in the East.*

² Twenty years previous to this, the treatment of hepatic abscess, whatever it may have been, does not appear to have become famous for its success. One of the charges advanced against Count Lally was 'of causing himself to be treated as if he had an abscess of the liver, before an abscess was formed, which, had it ever happened, would have caused his death.'

³ Octavo edition, p. 500.

tually than we can. Consequently, to give her fair play, our treatment ought, to a very limited extent, to be expectant. Still, we must not stand passively by until the disease has become hopelessly extensive and the patient has lost all reparative power.

Exploration for Hepatic Abscess.

Writing in 1854,¹ Waring showed that the exploratory needle, employed for this purpose, was in use many years antecedent to that date. It was first brought prominently to notice by the late Drs. Mouat and Murray, in 1839. Dr. Murray, as cited by Dr. J. Cameron, stated that the Indian *hakims* had 'immemorially resorted to puncture of both liver and spleen for the dispersion of enlargement with induration.'

The needle was three or four inches long, with a canal instead of a groove. Twenty-four cases are given in which this instrument was used. Among these, we find such entries as the following occur: 'Exploratory needle, introduced its full length into the liver; no pus flowed, no bad symptoms followed; on the contrary, the patient seemed to feel better.' 'Exploratory needle used. A small quantity of serous fluid drawn off—afforded relief.' Then again: 'Trocár only employed. Pushed in its full length. Blood only escaped; no pus; afforded great relief.' Upon these cases, Waring remarks that they tend to show that the mere fact of exploring the liver is unattended with imminent danger or any immediate ill-consequence.

In the treatment of my own cases, I did not use exploration, except where adhesion and pointing had occurred, although I felt assured that many of my brother officers employed it with great judgment and success. Writing in the '*Lancet*' in 1863,² Deputy Inspector-General Dr. Cameron said, 'I have repeatedly plunged a trocar deep into an enlarged liver without finding an abscess, and never had the slightest ill-result beyond a little local pain, yielding readily to opium, or a few leeches round the puncture. I have also found such puncture followed by gradual absorption and disappearance of the enlargement. In men who have died with abscesses that I failed to reach, I have found it extremely difficult to detect the marks of such unsuccessful operations, and this, too, where there have not been any adhesions.'

I have now before me a beautifully delicate trocar and canula, five and a half inches long, which was given to me by my eminent friend Dr. Brougham, of Culduthel, who often used it freely without any ill-effect. He, like his predecessors, is convinced that, even when pus is not found, the escape of blood from the engorged liver appears to give decided relief. I argued, perhaps wrongly, that, as I have always had a great dread of being the immediate cause of a patient's death, and as I should not have submitted to such exploration in my own case, I

¹ *Op. cit.*, p. 181.

June 6 and 13.

would not recommend it to my patients. Nevertheless, I cannot for a moment deny that, in certain cases, exploration is advisable, and I cannot find any instance in which the use of the exploratory needle or of Brougham's fine trocar has done any harm whatever.

My colleague and friend Dr. William Palmer made the following communication to Sir Joseph Fayrer: ¹ 'You have asked me to give a brief account of the results of treatment by puncture in cases of doubtful liver abscess which came under my treatment during the period of six years, in which I had medical charge of an average of about seventy patients in the European General Hospital, Calcutta. Passing over cases of undoubted liver abscess, there was still a residuum of patients presenting doubtful symptoms, in whom neither unsymmetrical enlargement nor superficial tumescence, &c., could be detected. Such patients presented doubtful symptoms varying in every degree; at the one, extreme cases of general cachexia, with irregular slight febrile attacks, would exhibit symptoms as frequently attributable to deranged stomach, or bowels, or lungs only, as to the liver itself; while, on the other, slight general enlargement of the organ would be found associated with that peculiar form of "tenderness" in which pressure over the organ produced an indescribable sensation, inducing either faintness, hurried respiration, palpitation, or nausea with retching, or all of these at once.

'In all of these cases, it was my custom to plunge a long trocar and canula, of small diameter, into any or all parts of the liver, through a valvular opening, examining on the spot a small quantity of extracted matter for pus globules.

'It was only in very exceptional cases that any signs of pus could be detected. When it was so detected, the puncture was generally followed by slight inflammatory action at its seat, which probably ended in adhesion of the organ to the parietes, and so facilitated the future opening of the abscess. When, on the other hand, no pus was found, a good deal of anxiety was felt, in the earlier cases, lest the puncture should be followed by any evil results. Such moments of anxiety, however, ceased to recur, for it very rarely happened that the patient did not express himself the next day as feeling very much relieved; and in no case do I remember any bad consequences resulting from such punctures. The relief was frequently only temporary, in which case a second, a third, or a fourth puncture was made at intervals of eight or ten days. In some, however, one puncture sufficed to cure.'

Lately, the practice of puncturing the liver, with a view to reducing congestion and tension, has suggested itself to Dr. George Harley.²

Reading only thus far, the young practitioner might infer that 'prospecting,' exploration, and local phlebotomising in hepatitis and liver

¹ Fayrer's work, cited above, p. 218.

² *Vide* chapter on 'Hepatic Phlebotomy,' *op. cit.*, pp. 161 and 509.

abscess have long been an established practice unhesitatingly approved of by the best authorities. A little research will, however, show that, since its revival in 1839, it has undergone great vicissitudes of censure and praise. When Dr. Cameron's paper, cited above, appeared in the 'Lancet,' Professor Maclean of Netley wrote¹ in strong disapproval of 'prospecting' for pus in the liver. He 'was present at a great many of Dr. Murray's explorations in cases where the abscess was a matter of "faith," and more legitimate operations where it was a matter of "certainty"—and he remembered the single case, of a soldier of the 57th Regiment, that had a fortunate issue.' He adds: 'For more than ten years, I was Residency-Surgeon at Hyderabad, in the Deccan; and, during that long period, had an immense field of observation before me. I gave this operation of puncturing hepatic abscess a very patient trial in native patients, without a fortunate issue in a single example.' His conclusion was: 'It is hoped that what has been advanced in this communication may have some effect on the minds of young physicians, teaching caution in surgical interference, except in cases of abscess clearly and unequivocally pointing to the surface.' The data were reviewed by Dr. Cameron in the 'Lancet' of August 8, and the whole question was summed up at page 224 of the 'Annals of Military and Naval Surgery,' by an Indian surgeon of great experience and admirable discriminative power, who—having cited the opinions of Dr. Budd and Sir Ranald Martin which were adverse to operative interference and in favour of leaving the evacuation of the abscess to nature—considered that 'the question is very fairly stated by Sir Henry Cooper, M.D.,² who wrote: 'The proposal to leave the opening entirely to nature is undoubtedly sound and good, provided the powers of the patient are adequate to the long process of ulceration through the textures. But this is assuming a greater amount of vital power than is usual at this stage of the disease; and it must not be forgotten that the patient is thus exposed to the constant risk of spontaneous rupture.'

It will be borne in mind that, in dealing with the 81 cases operated upon of whom Waring tells us 15 recovered and 66 died, surgeons, who were perhaps a little enthusiastic in favour of a new practice, employed coarse instruments—the large ascites trocar and that in the hydrocele case—both of which I have used only to be dissatisfied with them—and a 'flat' trocar which I never saw. They had not Brougham's fine but valid trocar, or Fayrer's aspirator, while they were not aided, in diagnosis, by the clinical thermometer, and, in after treatment, by the antiseptic system.

I never attempted to evacuate the pus until actual adhesion and pointing occurred—that is to say, until adhesion of the serous surfaces

¹ *Lancet*, July 18, 1863.

² *British Medical Journal*, May 23, 1863.

was manifested by infiltration and inflammatory redness of the integument. Attempts used formerly to be made, especially by French surgeons, to obtain adhesion of the serous surfaces by incision and caustics. These have, I believe, long been abandoned; one of the greatest dangers in liver abscess being sphacelus of the integument. Here it is to be remarked that, if we lose any time unnecessarily in opening an abscess, the tendency of which is to point externally, the integument will become undermined by burrowing pus, and will almost inevitably slough. This *gangrene of the undermined integument*, which is frequently attended with *necrosis of the ribs*, is one of the deadliest accidents to which the victim of liver abscess is liable.

I know of only one such case in which recovery occurred—Sir Joseph Fayrer's case of a native, Case 7.¹ Those who used to operate by a large opening, when the surfaces were not adherent, generally used the trocar and canula—leaving the latter in the wound. In the absence of adhesion, the operation has caused escape of pus into the abdominal cavity. Waring gives two cases of this unhappy accident. In one of these, the lancet was employed; in the other, the instrument used is not specified. I know of a third fatal case. I must repeat that I have never heard that the very slightest harm has been caused by the use of the fine instrument recommended by Drs. Brougham and Palmer, but quite the contrary. In this instrument, the principle of Dr. Pancoast's 'therapeutic knife' was anticipated many years ago.² By what he terms 'the antiphlogistic touch' of a very small knife, which leaves no scar, he punctures tumefactions of almost any sort in numerous places. By this, the best means of deep-seated blood-letting of the part, he can, if he sees the patient early enough, almost assure him against the formation of abscess. When a bubo or other gathering becomes hard and refuses to yield to local applications, he freely punctures it with the little knife, on the point of which he occasionally finds a drop of pus. As I have already mentioned (p. 611), I used this principle, in rendering my own boils abortive, twenty years ago. Waring, agreeing with the late Drs. Mouat and Murray, justly observes: 'That the evacuation of these abscesses at an early period is desirable and advantageous, is an opinion I entertain in common, I believe, with most of my medical brethren in India.'

In like manner, Mr. Hare has insisted: ³ 'I feel certain that a very large proportion of patients with abscess of the liver, who now die by long neglect of the operation, might be saved by a little boldness.' My own experience convinced me that many deaths occur because the operation has been too long postponed. We have seen that out of 81 cases, collected by Waring, in which the contents of the abscess were evacuated

¹ *Op. cit.*, p. 211.

² *Philadelphia Medical News*, October 24, 1885, and *M. T.*, vol. ii. p. 794 for the same year.

³ *I. A.*, No. 18.

by operation, there were only 15 recoveries, or 18·519 per cent. But these cases occurred when bleeding and mercurialisation were in use, and before the employment of antiseptic dressing was introduced.

I have used the fine aspirator, the small (hydrocele), and the large trocar. In a native man, who had pointing between the ribs low down, I employed the fine aspirator. About five ounces of salmon-coloured pus, free from tissue shreds, escaped, and he recovered quickly without a bad symptom. This was my only complete success with the aspirator. In other cases where I used it, the small tube always became clogged with broken-down liver tissue. Annesley insisted upon this objection to the use of the trocar. In April, 1880, Sir Joseph Fayrer showed, at a meeting of the Medical Society of London, an aspirator fitted with long and large trocars which appeared to be admirably adapted for the free emptying of these abscesses. It is now the rule to treat all these cases antiseptically.

I have spoken of *Kirrrosis* under the head of Malarial Cachexia (pp. 232, 253). European and native drunkards are liable to it, but I believe that this condition is rarer in Calcutta than it is in London. I do not think that gin-drinking prevails in India.

Dr. Ewart has, in his Catalogue, placed the following notes against two specimens from natives in the M. C. M.

In 479, 'the hob-nail character of the surface is well demonstrated. The increased thickness of Glisson's capsule from organised albuminous exudation, converting the organ into a series of cellular provinces, is everywhere observable. It is, in fact, a perfect exemplification of degeneration and almost destruction of the parenchyma or secreting structure of the liver in a case of advanced cirrhosis.'

480. Dr. Webb wrote of this: 'The parenchyma of the liver is converted into a mere web-work resembling anasarcaous cellular tissue; the cells are all empty, and there is not a vestige of glandular structure left; there is nothing but Glisson's capsule. From a native woman.' Ewart notes: 'Here again the degeneration and destruction of the secreting structure of the liver has followed hob-nail contraction or cirrhosis.'

The occurrence of advanced kirrhosis in native women who have suffered from malarial cachexia but did not appear to have been drunkards, recalled to my mind a trick which was said to have been played upon a London pathologist who always called hob-nail liver 'gin liver.' One took to him a portion of the kirrhosed liver of a deer, and was told that it was a typical specimen of 'gin liver.'

Fatty Liver is developed in India nearly as in England. For Abscess in Fatty Liver, see p. 621.

Primary Cancer of the Liver is a rare disease in India. Morehead relates one case, in a native. I saw, with Sir Joseph Fayrer, the case of a young German woman, in which proliferation went on with such

remarkable speed that it was probable that the disease originated in India, although she had not been long in the country.

Dr. A. Porter has related¹ a very well-marked case, occurring in a Mohammedan prisoner, *æt.* 50, in whom primary cancer of the liver resulted in secondary deposit in the intestines and pleuræ.

Dr. Joseph Ewart has given² the following description of a specimen of primary cancer of the liver in our Calcutta Museum: 'The liver of a native female, about 45 years of age, under the care of Dr. C. R. Francis. She had been complaining for about four months. On admission, she was deeply jaundiced. The liver is 10 inches in length, 5½ broad at its left lobe, and 7 inches at the right. It weighed 5 lbs. 8 oz. It is studded throughout with nodular masses of medullary cancer. The cancerous cells appear to have infiltrated the hepatic tissue rather than to have commenced and maintained themselves in restricted independent provinces. Even in the smaller accumulations which, to the unaided eye, seemed quite isolated from and independent of the liver structure, there were found, mixed up with them, original biliary cells, and the vascular and duct structure of the lobules all more or less altered. The circular, oval, and elongated fusiform and endogenous cancer cells, and the free nuclei, were highly tinged with cholepyrrhin. There was no vestige of cancer in any other organ of the body. The patient had the cancerous cachexia fully developed. The existence of nodular prominences on the surface of the organ, with their rapid enlargement and that of the general mass of the organ, rendered the diagnosis easy during life.'

An aged retired Indian medical officer had jaundice which, on passing gall stones, was much lessened but did not disappear. He died with primary cancer of the liver.

Acute Atrophy of the Liver.—When Dr. Ewart published his Catalogue in 1865, the Collection at the Calcutta Medical College did not afford an illustration of this condition; but, in January 1868, Dr. Collis reported that Dr. J. F. N. Wise had sent them a specimen.

Prep. E 19 in Mr. Knapp's Catalogue of the Grant M. C. Museum, Bombay, is 'Acute atrophy of the liver. From a patient in the J. J. Hospital.' These two rare cases lay far apart—Dacca and Bombay.

Although I looked for it steadily, I am not certain that I ever saw this disease. I had a case in which the symptoms, ending in the patient's sudden death soon after his admission to hospital, led me to suspect the existence of this condition; but I could not obtain an autopsy.

Dr. G. C. Roy has recorded a case, the diagnosis of which he admits is an open question.³ A Hindoo man, *æt.* 40, had suffered from diabetes, with urine of s. g. 1030. He had come off a long journey

¹ *I. M. G.*, vol. iv. p. 190.

² *I. A.*, No. xxi. p. 309.

³ *I. M. G.* for May 1880,

and had suffered from mental anxiety. Had pain in the back. On the 4th and 5th day of his last illness, had sudden profuse epistaxis, followed by suppression of urine. On the 6th day, his mind began to wander. On the 8th day, he was drowsy, but could be easily roused, and then went off in stertorous sleep. The eyes were yellow; it is said that his relatives noticed 'yellowness of the upper half of the body.' There were subsultus and low muttering delirium. Reduced area of liver dulness. About an ounce of urine was obtained by catheter; it was albuminous, but no leucine or tyrosine was detected. An autopsy could not be obtained.

The Catalogue of the Madras Collection gives two cases of acute atrophy of the liver by Mr. Branfoot and Dr. W. H. Harris.

Prep. 121. From a Gentoo woman, *æt.* 35, admitted comatose, but could be slightly roused by shouting, though not sufficiently to enable her to speak. Conjunctivæ deeply jaundiced; pulse rapid, small and thready, hardly to be felt at wrist. Surface of the body cold, stools constipated, devoid of bile; urine scanty. That which was drawn off was dark coloured, contained one-fourth of albumen, sp. gr. 1016. Nitric acid turns it green. A light yellowish deposit in large quantity on standing. Liver dulness only a slight line, one inch in breadth, between sixth and seventh ribs. The liver weighed eighteen ounces. Its measurements were: right lobe, transversely, six inches; vertically, five and a half inches; extreme thickness half an inch. It was shrivelled and flabby, lying against the spine, capsule puckered loosely, pale in colour. The left lobe has almost disappeared; gall bladder nearly empty, containing two drachms of green, thick, and sticky mucus. In its greater part, the surface of the gland is greyish in colour; but here and there, it is mottled reddish-yellow, with patches of the same colour. On section, the cut surface is rhubarb coloured, but, near the circumference, the section has a pale greyish colour, and tracts of this grey colour run through the organ and round the lobules. Intra and sublobular veins full of blood, appearing as dark small spots; portal channels empty; bile ducts apparently empty. The microscope shows an almost total destruction of liver cells. In parts, no cells found, but an enormous number of nuclei and dark small granules. In other parts, liver cells are present, irregular in shape and filled with nuclei; there are also a number of fat cells [?]; these vary much in size, some being very large and bear annular markings (leucine), others refract as oil cells. No needle crystals of tyrosine found in blood or in section.

161. Acute atrophy of the liver. The organ weighed 34 tolahs.¹ A woman was admitted in the 7th month of pregnancy, complaining of giddiness, head-ache, and pain in the right hypochondriac region.

¹ The tolah is the weight of the rupee—180 grains.

On the following morning, she was almost completely unconscious, but, when roused, seemed to have hyperæsthesia all over the surface of the body and some muscular rigidity of the limbs. The conjunctivæ were somewhat yellow, but not deeply tinged. Urine was high-coloured, but not to an extreme degree, and contained the bile acids. Bowels constipated for three days. On being moved by an aperient, the stools were destitute of bile. The fingers could be inserted deeply upwards under the margins of the right ribs, and percussion yielded a clear resonant note all over the hypochondriac region below the breast.

Full reports of Indian cases of Acute Atrophy of the liver, *with particular notice of the state of the urine and of the condition of the renal structures*, are very needful. I shall recur to the subject of this disease in speaking of Jaundice.

Jaundice.

Writing in 1888, Dr. Edward Goodeve observed,¹ 'Simple jaundice, without much disorder of the liver, has attacked a large number of persons' [in Calcutta] 'during the spring of the present year.'

Upwards of eighteen years ago, a healthy girl of four was brought from Calcutta to Cheltenham. Shortly after her arrival, it was observed that she was jaundiced. This did not cause surprise, as the season (September) was very chilly and she had suffered much from heat and fatigue on the overland route. It was then noticed that her brother, an equally healthy child, three years her senior, who had been more than two years at home, was also jaundiced. The attacks were unattended with evidences of constitutional distress and were very transient. The only circumstances which these children appeared to have in common were that they had both eaten the same food, and had lived together for a very few days in the same house in unseasonably cold weather. I have heard of similar outbreaks of simple jaundice among the children of a neighbourhood, but am not aware that their occurrence has been explained.

The statistics of jaundice among the European barrack children during the four years ending 1883 are somewhat remarkable—5 admissions, no deaths; 9 admissions, 4 deaths; 6 admissions, 2 deaths; 5 admissions, 2 deaths. If, in the majority of the cases of this gravely mortal jaundice, the illness appeared during the hepatic crisis which immediately follows birth, the matter is plain. Otherwise it needs explanation.

In tropical climates, it is very necessary and is sometimes of great practical importance to distinguish from jaundice the sallow, dusky, yellow-greenish discoloration of the skin, the result of blood disintegration, which characterises Indian Relapsing and some types of Remittent Fever.

¹ *I. A.*, No. 1, p. 260.

A medical colleague of mine was under my care in Calcutta in a dangerous attack of remittent fever. He was very low, there were long-continued insomnia, an icteritious appearance especially of the face, and very pale urine of singularly low sp. gr. There had been dysenteric symptoms which had passed off. It was not a case of true enteric fever. Two of our medical brethren came one afternoon and told me that I ought to give a brisk dose of Epsom salts, which would probably remove the jaundice and give him sleep. I objected that I did not consider that the suffusion was jaundice, and that the tendency to dysentery forbade drastics. I believe that, had I acted upon this well-intentioned suggestion, England would have lost a man whom I hope to see acknowledged as one of her ablest physicians.

In many cases, jaundice obeys, in India, the laws which govern it in Europe. A few noteworthy facts have occurred in my experience.

A well-conditioned-looking European, about forty years of age, was seen in my ward at my morning visit. He had jaundice which was recent and very marked. He did not appear to be at all severely ill, but the urine was merely a little high-coloured. That afternoon, he fell into a state of eclampsia and died before I could see him. I found that his kidneys were in a very marked condition of Bright's disease. There was nothing in the liver that appeared to account for the jaundice or for the sudden death. I heard of more than one other case (one was in a young medical officer) in which acute jaundice was suddenly fatal, with head symptoms. Contrast these with the following not uncommon case.

A middle-aged European was in my ward, almost of the colour of a Seville orange. He had got dead drunk at night on the flat roof of his house, and had remained lying there fully exposed to the beating of an unusually heavy tropical storm. In the morning, he was jaundiced. He evidently had not the very least sensation of illness and, at every visit, desired to have beef-steak. He was so peremptory in his wish to go into town daily, that I had to remind him that the hospital was not an hotel. He recovered without a single bad symptom, but I have never seen urine more deeply coloured by bile than his was. Notwithstanding his evil habit, his kidneys were still sound enough to carry him through his danger. In practice, I considered the state of the kidneys in jaundice to be at least of as great importance as that of the liver. I would entreat every young medical man, as I did my students, most carefully to view every case of cholæmia and jaundice *in its renal aspect*. In the principal organic diseases of the liver (not including hepatic abscess) experience in the dead house gradually impressed upon my mind the axiom—*such as the liver is such are the kidneys*. This is especially the case in kirrrosis; and we have still, I think, much to observe in this direction.

I apprehend that, when jaundice occurs in a case of morbus Brightii, it is of necessity mortal. Doubtless, the comparative soundness of their kidneys accounts for the absence of general constitutional disturbance in certain subjects of hepatic cancer. I recollect an elderly man who appeared to be suffering from primary malignant disease of the liver whose complexion was spinach-coloured, but who was able to walk the streets with great activity. An aged brother officer, whose case is referred to above, suffered from gall-stone and excessive jaundice in which dangerous epistaxis occurred. I was not in attendance upon him, but, examining his urine, I told him that the evident healthiness of his kidneys was vastly in his favour. His illness was long, but he passed a very large calculus and then appeared to get nearly well, although considerable jaundice remained. Primary cancer of the liver was found at his death several months later.

I have before me the published report of a case which, although it occurred fifty years ago, still conveys an important lesson. The italics are mine. A European seaman was admitted to a Calcutta hospital for diarrhoea and fever. The spleen was enlarged. *There was tenderness, increased on pressure, over the right kidney.* As the case went on, it was reported that *the urine was scanty* and the extremities were cedematous. *Diuretic Purgatives* were prescribed. In the fifth week of treatment, jaundice appeared. *Blisters* were applied over the liver. Although *the urine was scanty*, it was high-coloured, tinging everything it came in contact with a bright yellow. 'A constant action of diuretic purgatives and counter-irritation over the liver were maintained.' On about the eighteenth day of jaundice, he 'had a severe fit of convulsions, fell into a state of coma and died.' The condition of the kidneys was so little regarded that these organs are not even mentioned in the account of the autopsy. I have no doubt that the diuretic treatment killed this man.

I fear that, up to the present moment, our students are not well taught in regard to the management of the kidneys in jaundice and in other conditions of hepatic difficulty. Although, as usual, Morehead's advice on this point is most cautious, it is, I venture to think, by no means absolutely safe. He says: ¹ 'As previously stated in reference to jaundice complicating remittent fever, saline diuretics are often given with great advantage; and, as a rule of practice in this disease, it should also be recollected that time is required for the removal of the bile from the blood, and that we may err by too great haste or too active interference.' Here the fine mind perceived light, but could not wholly shake off its old superstition.

Tanner recommended mild diuretics in jaundice from obstruction.

In this form of the disease Dr. Bristowe enjoins that the functions of the skin and kidneys should be promoted by the use of diluents,

¹ *Op. cit.*, p. 4.

diuretics, &c. In jaundice without obstruction, he would promote the action of the skin and kidneys.

Murchison enjoined that, 'in all cases of jaundice from obstruction it is important to maintain the action of the kidneys.'¹ In one obvious acceptance of his words, I quite coincide with this eminent authority, but his meaning is open to question. I asked a young physician how he interpreted this expression; he said—'we are told to give diuretics.'

If we say—examine the condition of the kidneys fully, relieve them, as far as possible, from all removable work, apply sinapisms and dry cupping to the loins if there be any evidences of Bright's disease or of renal congestion or embarrassment, keep the bowels open by laxatives, not by drastics, promote cutaneous action, but give no diuretic stronger than milk—we shall, at least, do no mischief in our jaundice cases.

I have already insisted upon the necessity for considering every case of cholæmia and jaundice *in its renal aspect*. This rule appears to have especial bearing in acute yellow atrophy of the liver. It is very desirable that the state of the kidneys in this condition should be fully investigated. True we are told that there is occasionally 'fatty change of the glandular epithelium of the kidneys,'² concurrent albuminuria has been repeatedly observed,³ and the presence of leucine and tyrosine in the urine is pathognomonic; still it remains to view yellow atrophy of the liver *in its renal aspect*, noticing all structural abnormalities in the kidneys, distinguishing between those renal lesions which are of old date and those which are due to the inordinate work which those organs have been forced to perform of late.

The cholæmia, head symptoms and hæmorrhages (discharges of hæmoglobin) which characterise acute atrophy of the liver point to failure of renal function. Pending further inquiry, the use of diuretics in this malady cannot be too strongly deprecated.

Remarking the intimate connection often found to exist in India between malarial fevers, scurvy, and jaundice, my friend Dr. Herbert Baillie advocated⁴ the free use of the juice of the ripe pine-apple in jaundice. The juice of other sub-acid fruits, such as oranges, grapes, lemons, and the pumplenose are serviceable, but not, as far as his experience goes, equal to the pine-apple. Under its influence, jaundice generally subsides in a week or ten days.

Sir Joseph Fayrer has recorded⁵ the case of a Swiss gentleman, aged 27, of stout frame and rather pallid and anæmic complexion, resident a few years in Bengal, during which time he enjoyed fair

¹ Quain's *ictionary*, p. 777.

² Bristowe, 4th edition.

³ Frerichs, Dr. G. C. Ray, *I. M. G.* for May 1880; Dr. Hyla Greves, *M. T.*, vol. ii. for 1884, p. 335.

⁴ *I. M. G.*, vol. v. p. 36.

⁵ *Ibid.* for December 1867, p. 260.

health, who met with an accident, while driving, occasioning compound fracture of the tibia and fibula. The evidences of constitutional shock were very marked and continued until he died at the end of forty-eight hours. Jaundice set in on the evening of the second day. The limb mortified. There was complete ischuria. There was no post-mortem examination. Sir Joseph Fayrer considers that the shock, which was most intense, acted on the nerve centres, causing such suspension of innervation in the ganglionic system as to induce jaundice and ischuria. He had previously seen jaundice as a result of shock, and also after capital operations.

Biliary Calculi occur in India nearly as they do in Europe, save that they are certainly far less common in grain-eating natives than they are in Europeans. There are several remarkably good specimens of biliary calculi in the Calcutta Medical College Museum, but the races of the sufferers are not specified in the catalogue. Early in my service, I had a case which occasioned me a good deal of anxiety and pain. A civilian of high rank, *æt.* about 62, was suddenly attacked with typical symptoms of 'gall-stone colic,' the suffering being most intense. His wife, a very resolute woman, told me that his disease was gout in the stomach, and requested that I would give one or two doses of calomel, a treatment which was considered to have speedily relieved him in several attacks of like severity. The case was not an easy one in itself. The patient was evidently one who had eaten and drunk 'generously.' There was gout in the family. It was denied that gall-stones had passed in the previous attacks. There was no jaundice. The stools were almost black, and loaded with undigested mushrooms. I see that, in his article 'Gall-Stones' in Quain's Dictionary, Dr. J. Wickham Legg says that 'some physicians think that the diagnosis should not be made unless the concretions be found in the stools,' and that 'it is agreed by nearly all that it cannot be made if there be no jaundice present.' But it is evident that, here, I had to make a diagnosis promptly and to act upon it forthwith. So much undue and imperative interference on the part of the lady, with whom I had no previous acquaintance, led me to say that, in my opinion, the case was not one of gout, and that large doses of calomel would do harm. The reply was that several medical officers, far more experienced than myself, had given this medicine with the best effects. The patient died with symptoms of effusion of bile into the peritoneal cavity. Examination revealed a solitary gall-stone, of a long oval form, almost as large as a hen's egg, occupying the gall-bladder, which, however, contained some healthy-looking bile. There was a very small ulcerated opening, on the under surface, just at the junction of the bladder with the duct. Through this, the fatal escape of bile had occurred.

Ten years ago, Dr. Joseph Ewart, of Brighton, and I, starting

upon the sixty hours' railway journey from Calcutta to Bombay, found that we had as a fellow-traveller a barrister whose career had been so eminent and successful that, before his retirement, he gained the position of a Judge of the High Court. As this gentleman had brought several spiritualistic treatises with him, our conversation turned much upon his supernatural experience. He told us that, walking in one of the northern suburbs of London, he was suddenly attacked with acute pain at the pit of the stomach, and sickness. A passer-by asked him to rest in his house, and suggested that his wife could obtain relief for him. The lady came, and the voice of one who was invisible was heard inquiring what his symptoms were, and prescribing a remedy. The voice, which was said to be that of a late Scotch physician, declared that his disease was 'inflammation of the liver and of the parts surrounding the heart.' Something having been done or taken at the spirit's advice, he slept, and awoke perfectly well. Upon my remarking that I had never met with a Scotch physician who was so absolutely ignorant of his profession, and that I was surprised that, in a higher state of existence, the late Doctor had not learnt more, the following dialogue occurred :—

'I value his opinion more than yours!'

'If you please; but such inflammation could not possibly have subsided in an hour or two.'

'Then what was my disease?'

'A gall-stone passed.'

'No, certainly not; at other times, when I have passed gall-stones, the symptoms were quite different.'

'Ah—then you are in the habit of passing gall-stones! I was not aware of that. Not one person in a thousand suffers from gall-stone. The symptoms always vary according to the size of the stone and other circumstances. You passed a gall-stone on that occasion!'

'I didn't!' (very indignantly).

Dr. Ewart—'You passed a gall-stone.'

In 1837, Dr. D. Stewart brought before the Medical and Physical Society of Calcutta¹ the case of a delicate female who, for seven or eight years previous to her death, had suffered from a large tumour in the right hypochondriac and iliac regions. It caused little or no inconvenience. She died of phthisis. The tumour proved to be an enormously enlarged gall-bladder which projected for some inches below the inferior edge of the liver. It was filled with a colourless fluid which was found to be pure mucus without a trace of bile. A small round gall-stone was impacted in the entrance of the cystic duct, which was quite impervious. The hepatic duct communicated freely with the duodenum and was considerably enlarged in diameter.

¹ *I. M. J.*, vol. ii., N. S., p. 562.

Prep. 474 in the C. M. C. Museum displays a by no means very rare condition of parts. A gall-bladder is filled with calculi; its parietes are much thickened and contracted firmly upon the contents of the viscus, which is strongly bound down by adhesion.

From such gall-bladders, the gall-stones not unfrequently make their way, by suppuration, to the surface.

When at Howrah, I saw with Mr. Burgess, an (East Indian) woman in whom a gall-stone could be felt through a sinus in the abdominal parietes. At that time, it appeared to be causing little or no irritation.

Dr. Herbert Baillie recorded¹ the case of an East Indian, *æt.* 47, who suffered from extreme and constant pain in the right hypochondrium, which was swollen. A free opening gave issue to a considerable quantity of healthy pus, which, after a few days, became of a reddish cinnamon colour, as if it came from the liver. This afforded great relief to the patient, whose health became much improved. About eight months later, Dr. Baillie found that there were two fistulous openings leading down into a cavity resembling the gall-bladder, from which fifteen small gall-stones, mostly faceted, and weighing a little over 100 grains, were removed by means of the finger and a scoop. The wound was entirely healed by the end of the fifth week, during which time there never appeared to be any admixture of bile with the secretions. Recovery was perfect.

Dr. James J. Green has recorded² the case of a Hindoo official, *æt.* 53 years, who had long suffered from intermittent fever at Chittagong. During one of these attacks, he was seized with acute pain in the right hypochondrium, where a swelling as large as a pigeon's egg appeared. In a few days the pain subsided, but the tumour remained, causing no inconvenience. Three years later, the tumour became painful and began to enlarge, and he had slight fever. The tumour was then three inches in diameter, partly above and partly below the edge of the ribs. On his lying down, it moved completely above the margin of the ribs. It was quite hard and circumscribed, a small spot alone in the centre being soft, and here there was distinct fluctuation. Caustic potash was applied to the soft spot; the result was the separation of a slough, and a moderate discharge of healthy pus. About a week later, pieces of gall-stone began to come away. Three entire stones and twenty-nine small pieces of gall-stone were discharged. The largest was triangular in form, with four facets on it, and measured half an inch in length and three-eighths of an inch in width. A small sinus remained, discharging healthy pus in decreasing quantities.

Dr. Waddell³ found, in an elderly Mohammedan man, the walls of the gall-bladder transformed into a dense bone-like cell, enclosing a large gall-stone.

¹ *I. A.*, No. 24, p. 295.

² *I. M. G.*, vol. i. p. 157.

³ *I. M. G.* for April 1884, p. 121.

There are several preparations in the M. C. M., Calcutta, illustrative of *obstruction to the gall ducts*. 464, partial obstruction of the common duct by the presence of a scirrhus tumour; 466, gall-bladder and common ducts atrophied and bound down by old peritoneal adhesions; 470, dilatation of gall-bladder from the presence of an enlarged gland upon the common duct, leading to more or less complete obliteration of its canal; 472, obstructed common duct with dilatation of the gall-bladder and bile-ducts.

Many years ago, before I went to India, I mentioned in the 'London Medical Gazette' the case of a woman in whom a gall-stone as large as a cob-nut was lodged half-way along the common duct.¹

Mr. Twining published a valuable series of cases of *occlusion of the biliary ducts* in the fifth volume of the 'Calcutta Medical and Physical Transactions.'

For the Distomata of India, see p. 26.

Diseases of the Pancreas.

Minute examination of this organ, pursued for a considerable time in every autopsy at the large Indian hospitals, would, I believe, throw important light upon the ætiology of many important diseases prevalent in that country, especially cholera, morbus Bengalensis (*vide* p. 565), sub-acute dysentery, hepatic abscess, malarial cachexia, and all conditions in which death is attributable mainly to failure of nutrition. Thus it would be well to know what the state of the pancreas is in the diabetes which is so prevalent in India.²

In my 'Medical Jurisprudence for India,' p. 563, I have cited in detail a very remarkable case by S. A. S. Suyd Mahomed Jaun, in which a native, *æt.* 50, having had his hands tied together over his knees, with a stick under his knees, was beaten and kicked until he fainted. *The Pancreas was vertically lacerated from above downwards at its right extremity through about half its breadth.* The whole organ looked highly congested, with extensive extravasation of blood into its cellular tissue.

In the remarks on abdominal wounds, p. 547, will be found a question regarding *protrusion of the pancreas* through such apertures.

A very remarkable case of *traumatic aneurysm of a branch of the Pancreatic Artery* is given under the heading of *Hæmatemesis*, p. 553.

Assistant-Surgeon A. Jayaker has reported a case of *Primary Cancer of the Pancreas*.³ An anæmic fakir was admitted with an apparently fluctuating tumour occupying the right hypochondrium,

¹ Unhappily, the printer made me say 'as large as a cocoa-nut.'

² See article on 'Pancreatic Diabetes,' *London Medical Record*, 1881, p. 150. *Medical Digest*, Section I. p. 336, cited in *I. M. G.* for October 1884.

³ *I. M. G.*, vol. v. p. 230.

epigastrium, and part of the left hypochondrium. He was anæmic and had diarrhœa. The case was supposed to be one of hepatic abscess communicating with the bowel. On exploring with a needle, only a few drops of blood escaped, which were regarded as being the only signs against the supposition that hepatic abscess existed. There was found a cancerous tumour of the pancreas weighing about five pounds. The liver was pressed far upwards; the other organs were compressed and anæmic, but otherwise normal.

In India, as in Europe, *malignant disease* of the pancreas and of the pylorus are found associated. In Dr. Waring's case of liver abscess, No. 151, the pancreas was greatly enlarged and hardened, pressing on the common duct and other ducts, so as almost to prevent the passage of bile.

Addison's Disease.

Dr. W. J. Moore has a note on this condition in his paper on Masked Malarious Fever.¹ He believes that 'morbid conditions of the supra-renal capsules sometimes, if not frequently, result as a portion of that general degeneration which we designate malarious cachexy. In two cases at least, *p.-m.* examination has shown enlargement (scrofulous?) of the supra-renal capsules, coexistent with malarious debility, spleen affection, and death by dysentery.'

We medical men of Calcutta were generally of opinion that one of our brother officers was a victim of Melasma Addisoni. He was a distinguished naturalist, over fifty years of age, for many years resident in malarial Calcutta. He was very reticent, even to his closest friends, on the subject of his health; but it was noticed, many months before his death, that he was markedly anæmiated and became gradually much emaciated. Although an almost total abstainer, he was a large eater, and was seen every day smoking in a small brougham with the windows closed. A naturally quick temper became, latterly, very sensitive; he was believed to have chronic diarrhœa, and the skin of his face and neck and of the backs of his hands was, latterly, intensely bronzed. His natural complexion was rather swarthy, but the melasma was very strongly expressed. He, at length, admitted that he was ill, and embarked for change to Singapore. When out at sea on a very hot day, he had been playing with a child in the saloon and retired to his cabin; at the time of the next meal, he was found dead in his berth, his unfinished cheroot having been carefully placed on a ledge beside him. There was no autopsy; but we all felt certain that he had died from syncope.

¹ *I. A.*, No. 22, p. 283.

DISEASES OF THE URINARY ORGANS.

Mr. Weir gives¹ the appearances in the body of a native lunatic in whom there was *congenital absence of the left kidney*. The right was much enlarged, and weighed 5 oz. 2 dr.

Preps. 827, 828, 829 in the C. M. C. Museum are modifications of *horse-shoe kidney*. There is no allusion to race. No. 829 was probably from a native.

It is noticed that, in each of these, there are two ureters, and that, in 829, the renal vessels are double. In No. 676 of Webb's Catalogue, the horse-shoe kidney was supplied by one common [arterial] trunk. Webb notices that the representation of a similar union of the kidneys was given by Bartholinus.

A case of horse-shoe kidney is reported in the 'I. M. G.' for 1855.

Prep. F 39 in the Grant M. C. M. is a specimen of horse-shoe kidney with two ureters.

Prep. 39 and 40, Series ix., in Sibthorpe's Catalogue of the Madras Collection, are 'Kidneys joined by a transverse band, and kidneys joined by a very short transverse band—Horse-shoe kidney.' 44 is a 'Horse-shoe kidney.' 'Each has a distinct ureter, but there is only one renal artery for both.'

In 1868, Dr. Collis received, at the C. M. C. Museum, 'A monstrous *Azygos Kidney* with double pelvis and two ureters.'² There is another case, probably imperfectly observed (one of the ureters having, I think, been overlooked), at p. 260 of the 9th number of the 'I. A.' Webb's Prep. 229 was 'Kidney having four renal arteries.'

There is notice³ of the case of a native of Ghazeepore who had *four kidneys and two spleens*.

Prep. 830 in the Calcutta M. C. M. is 'Left kidney of a' (native) 'female from the dissecting-room showing *two ureters*, which, however, are seen uniting into one before they reach the bladder.'

Mr. J. W. Johnston⁴ had under his care a trans-Indus Pathan, the subject of *movable kidney*. After a time, the organ ceased to wander by becoming firmly attached to the left abdominal wall, apparently by local peritonitis.

Intermittent Neuralgia of the Kidney.—My friend Dr. Robert Bird⁵ relates the case of a strong middle-aged European who was seized with neuralgia of the left kidney. This disappeared after a few hours, but recurred the following day and continued in the same manner to disappear and recur daily for a week. He was then attacked with

¹ *I. M. G.*, vol. vii. p. 272.

² *I. M. G.*, 1868, p. 83.

³ *I. M. G.* for January 1874, p. 11.

⁴ *I. M. G.*, vol. viii. p. 184.

⁵ 'Remarks on Malaria and some of its effects,' *I. M. G.*, vol. i. p. 55.

intermittent fever, and, on the two following days, he suffered from similar attacks of ague followed by fever. The disease yielded readily to quinine, and it was found that the fever had wholly displaced the kidney symptoms—as these did not accompany the fever nor did they return. A dull pain over the region of the kidney was complained of for some days after all the other symptoms had been recovered from, but gradually disappeared without treatment.

Dr. F. W. Kirkham has lately reported ¹ a very noteworthy case of an engineer who had been resident for eighteen months on the Isthmus of Panama, in whom, after two previous attacks of intermittent fever, there occurred daily agonising pain in the right loin, extending down the thigh and affecting the end of the penis. His condition much resembled that which attends the passage down the ureter of a renal calculus, save that the attacks were periodical, were attended with marked rise of temperature, ended in profuse perspiration, and yielded to quinine.

I believe that I saw three of these cases in Calcutta, which, in the absence of periodicity and marked fever, were less distinctly typical. The patients, an East Indian youth and two officers, recovered without evidences of spinal disease or stone.

Morbus Brightii.

I have already spoken of cases of *Albuminuria* under the heading of Malarial Cachexia, p. 242. Many natives and some Europeans become the subjects of the ordinary forms of Bright's disease consequent upon spirit drinking, but London gin, not being much used in India, other spirits, more common there, do not appear to act so injuriously upon the kidneys as that does, with its four diuretics—gin, juniper, white vitriol, and turpentine. Having had, at one time, large experience of London drunkards, I had formed an idea that a free and steady drinker, say the landlord of a London public-house, could, all other circumstances favouring him, live for about seven years before he died from hepatic and renal disease. But, in India, doubtless partly in consequence of the great freedom with which the skins of drunkards frequently act for years there, a life of drunkenness may be far more prolonged. I knew a European who drank steadily for fourteen years after his first attack of drunkard's epilepsy. But this man's surroundings were peculiarly favourable. He drank good liquor, had nothing to do, enjoyed competence, and had a wife who steadily endeavoured to save him from perishing. Only a few days before sending these pages to press, I learnt from the brother officer who succeeded to my unpleasant duty of attending to this person's health that his death was not, after all, the direct result of drunkenness. Changing his residence, he

¹ *M. T.*, vol. i. for 1885, p. 443.

fell ill and consulted a strange doctor, who gave him mercury, which caused death by salivation—a natural result in one who, settled for many years in a very malarious spot, had unswervingly ‘gone in’ for hepatic and renal disease.

Preparations 53, 54, and 58, Series 10, in the Madras Collection, illustrate *Lardaceous or Amyloid change of the Kidneys* in a European and two natives.

Cystic degeneration of the cortex is frequently seen in elderly Europeans and natives.

Prep. 813 in the M. C. M. is a ‘*very small granular kidney*, the cortical structure of which is greatly nodulated, resembling in appearance a hob-nail liver.’ I sent this; but I cannot recollect the patient, as I saw many such specimens.

Primary Medullary Cancer of Kidney.—Dr. R. D. Murray records¹ the case of a boy, *æt.* 6, with an English name, in whom there were two normal kidneys, whose death resulted from cancerous perforation of the cæcum, to which had adhered a *supernumerary floating kidney the seat of medullary sarcoma*. The bladder presented only two normal ureter orifices. What appeared to be the remains of the third ureter was lost in the cancerous mass. It appears probable that this canal had opened into the right ureter. The diseased organ measured $6 \times 3\frac{1}{2}$ inches.

I had under my care two Europeans suffering from *Medullary Cancer* of the kidney. In the rather slow course of their disease, I noticed an interesting fact of which I have not seen any mention. After long-continued large discharges of blood, these ceased in both cases, many days previous to death. This arrest of exhausting hæmaturia appeared to be a relief, but the patients’ health steadily declined. In both cases, a plug of coagulum had formed in the pelvis and had completely occluded the ureter. The renal artery and vein had become so contracted as evidently to act no more than the part of nutrient afferent and efferent vessel to the still highly vascular mass which, no longer retaining any vestige of gland-structure, had become a mere fungoid tumour. The other kidney, which was much hypertrophied, had worked well. There was no appearance elsewhere of secondary cancer.

The late Dr. Chuckerbutty published² the case of a stout, well-built European sailor, *æt.* 20, who, having drunk, after severe exercise, a large draught of cold water, felt chilly and sick, and then had aching of the limbs and rigor. On the following day, he began to suffer from acute pain in the right loin, extending from the crest of the ilium to the last rib. After remaining under treatment for nearly two months, he was discharged apparently cured. By a very careful line of argument, the reporter inferred that a *Perinephritic Abscess* found discharge by the neighbouring bowel.

Preps. 814 and 815 show *Renal Abscess*, but are without histories.

¹ *I. M. G.*, March 1886.

² *I. A.*, p. 269.

In the chapter on Liver Abscess (pp. 645-46), I have given cases in which the sac encroached upon the substance of the right kidney, and others in which renal abscess, probably ichoræmic, existed as a complication of hepatic abscess.

I attended, in consultation, a fine-looking elderly native merchant of Bombay, visiting Calcutta, in a fatal attack of *Ischuria*. He being a stranger, there was no satisfactory account of his previous condition (presence of albuminuria, &c.), and the body could not be examined after death. There is a report¹ by Mr. J. Bird of the case of an intemperate invalided soldier, *æt.* 50, who died of *Ischuria Renalis* on the seventh or eighth day of suppression. In the four years ending 1888, a European soldier, a prisoner, and four native soldiers were admitted for 'suppression of urine,' but the only death was that of a sepoy.

Since my return to England, I have seen cases of *Hæmaturia* in a young gentleman born in India and in an aged English official long stationed at Hong Kong. In both of these cases, there was a tendency to ignore *calculus lodged in the pelvis of the kidney* chiefly because there was not any lumbar pain; but, on sifting the aggregate of symptoms and circumstances, everything appeared to me indicative of this conclusion. A calculus fills the pelvis; in course of time, others form in the infundibula, the kidney ceases to act, its fellow works for it, its glandular structure wastes, and, at length, there remains little more than the proper capsule filled with calculi. It is singular that both kidneys may thus go on suffering and wasting *pari passu*. A parcel was sent to a Museum labelled 'Scirrhus of the Kidneys,' but without history. I found that both glands were in the above condition. It was difficult to understand how life could have been maintained where there was so little renal structure.

For *Hæmaturia* and *Hæmoglobinuria*, see Malarial Cachexia, page 245.

I have nothing to add to our present knowledge of *Lymphuria* (*vide* p. 265). Only three cases occurred in my own practice. I believe that the patient in whom Lewis first observed the parasite was repeatedly an inmate of my ward. The S. C. R., for the four years ending 1888, give only two admissions, probably of the same individual, a prisoner.

Pyonephrosis and Hydronephrosis.

In 1868, Dr. Colles received, at the Calcutta M. C. Museum, a specimen reported as a 'large abscess of the kidney with dilated pelvis and calyces.'

Dr. William Mortimer reported² the case of an Indo-Briton of Seringapatam, who had suffered severely for some years from the endemic fever of that station. On his admission to the Madras General

¹ *C. M. T.*, vol. iv. p. 20.

² *Madras Quarterly Medical Journal*, vol. ii. p. 64.

Hospital about a fortnight before his death, his belly generally was very tumid and tense; the left side rather more so than the right, but without any particular uneasiness on pressure or distinct fluctuation. He complained chiefly of debility with occasional difficulty in breathing, and loss of appetite. There was no apparent urinary complaint. He was very weak and emaciated on admission, and gradually sank.

An immense membranous sac presented itself on opening the abdomen, extending from the ensiform cartilage to the pubes, occupying the place of the small intestines. The transverse colon traversed it obliquely. It adhered to the diaphragm and to all the viscera with which it was in contact. Posteriorly, it was adherent to the blood-vessels and vertebral column. It contained upwards of a gallon of thin puriform fluid. Its interior was traversed by septa which divided it into several small chambers. The blood-vessels of the left kidney were not made out, but its ureter was found perfectly healthy as far as it could be traced. The sac was evidently the left kidney, of which no other trace could be discovered. The right kidney was enlarged and its ureter was prodigiously distended.

Mr. J. Lawder treated¹ the case of a Madras Pariah who had been long subject to intermittent fever in Mysore. About three months previously, he had been attacked with acute pain in the right side. From that time, the abdomen became generally enlarged. It was considered that there was an abscess of the liver, and an incision was made at a prominent point between the ribs and umbilicus. As it was found that there was no peritoneal adhesion, the wound was closed. Five days later, he was seized with symptoms of effusion into the peritoneum, but he lived, gradually sinking, eleven days longer. The abdominal cavity was found to be generally distended by a large sac, traversed obliquely in front by a part of the colon. From it 23 pints of 'pure pus' were drawn off. They 'found this immense sac to be the external coat of the left kidney thickened, and, except a small portion, the whole parenchymatous structure completely destroyed and the ureter obliterated. The bladder contained a pint of high-coloured urine.' As it is said that 'all the other abdominal viscera were perfectly healthy,' it is to be presumed that the right kidney was considered normal.

Although the contents were puriform in both of the above cases, these were probably examples of *hydronephrosis*.

Mr. R. D. Murray has reported² with great care, a case of *hydronephrosis* occurring in a native man of 60. The left kidney measured 7×4 inches and was $9\frac{1}{2}$ inches in circumference. It contained nine ounces of fluid, partly pus, partly altered urine holding cholesterine in suspension, and twenty-two faceted calculi. One of these (oxalic) was

¹ C. M. T., vol. vii. part ii.

² I. M. G. for 1878.

as large as a pigeon's egg and weighed two drachms. The others were small. The right kidney measured nine by six inches, and was twelve inches in circumference. Its weight was thirty ounces. It was reduced to a large multilocular cyst, the walls of which were membranous and semi-transparent. Two oblong calculi completely closed the ureter an inch down. It contained twenty-nine ounces of fluid.

Baboo A. C. Kostagree¹ admitted to the Burrisaul Dispensary a Mussulman, *æt.* 24, who appeared to be the subject of ascites. His belly was so distended that three strong men were required to move him from one place to another. He kept the sitting posture day and night, being unable to lie down; he dozed at night as he sat. The urine was scanty and high-coloured. Upon tapping four inches below the navel, nothing escaped from the abdominal cavity. Death took place three days later. The left kidney was small and atrophied. The right occupied the whole abdominal cavity. 'It was fatty in nature,' and contained six cysts full of urine. Without this fluid, the organ weighed 1 *maund* and 1½ *seers* (eighty-three pounds). Unfortunately, there is no allusion to the condition of the ureter.

Stone being of very frequent occurrence in India, *Calculous Disease of the Kidneys and Ureters* is often observed.

In 1867, Dr. Collis reported that he had received from Hazareebaugh a 'kidney converted into a multilocular cyst, from obstruction of ureter by a calculus;' and from Monghyr, 'occlusion of ureter by a calculus, and conversion of kidney into a mere cyst, with hypertrophy of the sound kidney.'

Prep. 832 in Ewart's Catalogue is justly characterised as 'a very beautiful specimen. An oval calculus quite fills up the cavity of the contracted bladder, so hypertrophied that the thickness of its walls varies from a half to three-quarters of an inch. There is a calculus fully an inch in diameter in each ureter. The whole of the glandular substance of the *left* kidney has been destroyed—the organ presenting a huge pelvis, with many sacculations replacing the parenchyma. The secreting portion of the *right* kidney is hypertrophied; but the increasing size of the pelvis has begun to promote its destruction.'

Geographical Distribution of Calculous Disease in India.

A thorough investigation of this question, throughout India, with especially full observation of Locality, Soil, Climate, Water, and Food, is a very needful undertaking. It would probably lead to the attainment of highly valuable data, particularly with regard to the causes and prevention of urinary calculus, which, at present, occasions a vast amount of suffering in many large districts. The illustrations of this question which I have been able to collect are few but very suggestive.

¹ *I. M. G.*, vol. ii. p. 12.

Thus—taking a survey of the British possessions from east to west, we at first meet with—

Districts in which Calculous Disease is absent or rare.

Mentioning to my friend Dr. Waring that I did not find any allusion to stone in his account of the diseases of *British Burmah* which is often cited in these pages, he said, ‘Stone does not occur there.’ The table at page 678 shows that only one case of stone was reported among the prisoners there in the four years ending 1883. This was in the year 1880, when the average number of prisoners was 4,656 and the admissions were 4,055. This solitary individual may have been a stranger. Rice is the staple food of Burmah.

Lower Bengal.—The rice-eating inhabitants of the alluvial Delta of the Ganges are almost free from liability to stone in the large eastern district of *Chittagong*. Mr. Bedford, my predecessor in medical charge, had, I think, one case of stone (probably in a stranger) in four years. In three years and a half, I never saw or heard of a stone case. My successor, Surgeon-General W. B. Beatson, has just written, in reply to my inquiry,—‘During the nine years I was civil surgeon of Chittagong, I never had a case of stone.’ He thinks that a little boy came to the dispensary with a calculus impacted in the orifice of the urethra. ‘I am,’ he writes, ‘quite certain that I never cut for stone till I left Chittagong. At Tipperah, I believe they used to have occasional lithotomies. At Dacca, I had, in nearly two years, 12 lateral lithotomies and one operation for stone in a woman. The cases came, I believe, from neighbouring villages, mostly to the north. At Nagpur, stone was most frequent, and I performed lateral lithotomy 53 times in the course of the eleven years I was there, and other cases were operated on by the sub-assistant surgeon. Still, I suppose ten or twelve cases per annum counted all, while at Raipur, 160 miles distant to the east, they used to have, I believe, 200 or more a year.’

Again, with regard to the statistics of stone in the eastern districts of Lower Bengal, so rare were lithotomy cases in his large circle, which included the extensive districts of Assam, the Cherra Hills, Sylhet, Mymensing, Chittagong, Tipperah, Nowakolly, and Burrisaul—in fact the whole of Eastern India—that, when I served there thirty-seven years ago, the superintending surgeon of the *Dacca* circle kept a case of lithotomy instruments, which the medical officers were desired to write for on occasion.

The Mitford Hospital, at Dacca, was not established at that time. Mr. Cutcliffe and Baboo Tara Prossunno Roy showed that, between the years 1858 and 1869, there were 166 lithotomy cases in the institution. The situation of Dacca, on a great river, renders it compara-

tively easy for resort from immense tracts of country, widely differing in soils and products. This table does not show whence the cases came, save that Mr. Cutcliffe's own eleven cases came from the following districts: Tipperah, *one*; Mymensing, *four*; Dacca, *six*.

A good many cases of stone present themselves in *Calcutta*—certainly more than used to appear previous to the opening of easy communication by rail between that city and the stone districts. Still, the stone cases which occur in *Calcutta* are very few as compared with the numbers received at such places as Raipur and Saharunpore. Thus: Fayer shows¹ that there were 50 cases of lithotomy in his wards in the M. C. Hospital, *Calcutta*, in 11 years, there probably having been about an equal number of cases in the second surgeon's wards. Thirty-eight lithotomy operations were performed by the late Mr. Allan Webb and by Dr. Herbert Baillie, at the *Calcutta Native Hospital*, in the years 1863–4–5.² I have already mentioned that Mr. Partridge had 20 lithotomy cases at the *Calcutta M. C. H.* in 21 months.

I am anxious to impress upon future observers the fact that—to render a case of stone, presenting itself for treatment in Lower Bengal, useful, as showing the comparative prevalence of calculous disease there, in the North-Western Provinces, and in other parts of India,—we should ascertain where the disease was contracted. Thus, I do not know whether the large majority of the *Calcutta* lithotomy cases, of which we have statistics, were Bengalis or up-country men. There is always a large number of the latter to be found in *Calcutta*. The record of an Indian stone case will always remain deficient in most important particulars if it does not contain information as to not merely the locality from which the patient last comes, but the places of his birth and bringing up, his race and caste, and the staple of his diet—especially whether this is *atta* (wheat), *dall* (lentils), or rice. Morehead insisted that 'it is very desirable that the place of birth and residence, varieties in modes of life from habits of caste or difference of circumstances, should be inquired into and recorded.'

Madras.—It is extremely noteworthy that we find, in the Annual Report of Civil Hospitals and Dispensaries in the *Madras Presidency* in 1883, that only two cases of 'removal of calculi from the bladder and urethra' occurred in 1,693,809 patients treated. These are apparently the two cases of Hindoos upon whom Mr. Maitland performed lithotomy in the General Hospital. When I mentioned this the other day to Surgeon-General Balfour, whose experience in that Presidency extended to every district, he said, '*Madras* is exempt from stone.'

During eleven years' practice in *Travancore*, Dr. Waring never saw a case of stone. The people live upon rice. No wheat is grown there.

The returns show that, in the four years ending 1883, not a single case occurred among the European troops stationed in the *Madras*

¹ *Observations*, p. 385.

² Mr. Reilly, *I. M. G.* for May 1866.

Presidency; and, within that period, only twelve sepoy and one prisoner were treated for stone. It is not at all certain that the whole of these thirteen individuals contracted the disease within the limits of Madras Proper.

Bombay.—Calculous disease, of indigenous origin, must be rare in the city and in the adjacent districts. When Morehead wrote, in 1860, the Grant Medical College Museum, Bombay, contained only 50 specimens of urinary calculi. The number of operations for lithotomy to which he alludes as having been performed by Bombay surgeons is comparatively small. Thus, by Mr. Bhawo Dajee (who, I believe, practised almost exclusively in the city of Bombay), *eleven* cases. Mr. Sadasheo Hemraj reported *three* cases from Bhooj. 'In the Catalogue of the Grant College Museum, there is an analysis by Dr. Watson of *nine* calculi extracted by Dr. Ritchie at Mooltan.' Although under the Government of Bombay, Mooltan is in the far north of that Presidency, and is in the stone district. The Catalogue shows that, in 1879, this museum contained 119 urinary calculi. Dr. H. V. Carter mentions that nearly one-half of these specimens came from the Jejeebhoy Hospital, being, he supposes, nearly every one extracted by the surgeons during a period of 11 years. This is not many in a city which, forty years ago, contained nearly six hundred thousand inhabitants, and is a large port, the terminus of the western highway from the stone districts. In Dr. Leith's 'Deaths in Bombay,' which bear every mark of having been drawn up with great care, and contain many valuable facts, I find, in three out of six years ending 1859, no mention of urinary calculi, while, in the other three years, there are only four entries: two of stone in the bladder, one prostatic, and one renal.

Dr. John Craig, who is well acquainted with *Scinde*, tells me that he was, for two years, in charge of the Native General Hospital at Bahawulpore, on the Sutledge, and only had one case of stone in the bladder.

In the subjoined tables we find that the cases of stone in this Presidency, during the four years, were: European soldiers, none; sepoy, 14; prisoners, 2. No case in jail in the *Berars*.

The above facts establish a law to which I know no exception—that *the rice-eating native peoples who inhabit the maritime face of India are exempt from stone*.

Districts in India where Urinary Calculus is very prevalent.

The principal of these are: the North-Western Provinces, the Punjab, and Central India. The four years' tables give, for the whole *Presidency of Bengal*, including the stone districts:

European soldiers, 8; native army, Bengal, 30; Central Indian force, 5; Punjaub frontier force, 21; prisoners, 34.

These figures, although perfectly accurate as regards the large classes of persons reported upon, give no idea whatever of the vast amount of stone which occurs among the poor of the civil population in certain districts. The following data set forth, to some extent, the prevalence of calculous disease in Upper India, from east to west, and thence north-west, at the foot of the hills in the North-West Provinces and the Punjaub; again from west to east in Central India. Tracing these names on the map, it will be found that every locality which is notorious for stone is one at which patients travelling from districts skirting the great mountain ranges are likely to apply for surgical relief.

It will be noticed that many of the dispensaries, at which stone cases most abound, are situated far down in the plain country; but it will also be generally observed that no stations at which civil surgeons are posted lie between them and the hills. If we sifted the stone cases received at any given dispensary, we should generally find that they do not at all represent the prevalence of calculous disease in the immediate district, but that of an exceedingly wide-spread area, which, at some point, touches the hills.

When my friend and colleague, Dr. William Palmer, was first stationed at *Ghazeepore*, he saw very few cases of stone. Gradually, more of these patients came in. He, at length, operated upon upwards of 200 patients; having, on an average, about one lithotomy a week. These were not, for the most part, inhabitants of the *Ghazeepore* district, but had generally travelled long distances from places at the foot of the Himalaya.

Hence, although a surgeon having a Government dispensary up country may generally obtain, as soon as he acquires wide reputation as a successful operator, a larger number of stone cases than he could expect to meet with anywhere else in the world,¹ it is probable that urinary calculus is not quite so prevalent in India as, at the first sight of its large statistics, it appears to be.

Between 1847 and 1855, the late Dr. A. Simpson extracted 188 calculi in the districts of *Mynpoory*, *Sarun*, and *Tirhoot*, in plain country, due south of the Khatmandoo Hills. Mr. Durant performed 21 lithotomy operations at *Tirhoot* (Mooteeharee) between July 1855 and December 1857.

At *Goruckpore*, stone is very remarkably common. At *Benares*, westward of these stations and very considerably south of the Himalaya, but on the great river and main railway line, my friend Surgeon-

¹ Mr. Holmes mentions that a surgeon told him that, in the North-Western Provinces, he had performed as many as eight stone operations in one day.

General Robert Cockburn had not more than 12 lithotomy cases annually; although there, at Saharunpore and at another sub-Himalayan station, he had about 150 lithotomies. Rai Ram Narain Das, Bahadur, cut for stone, at the *Cawnpore* and *Budaon* dispensaries, 248 times in 12 years. While he was absent from the latter station for seven months, 37 cases were operated upon by the medical officers who acted for him.¹ Sub-Assistant-Surgeon K. C. Nundy operated, at *Goorgaon*, upwards of 160 times in six years.

Principally at *Meerut*, the late Mr. H. C. Cutcliffe performed 111 lateral operations.² Mr. Thomas C. Heathcote, whose valuable life was early cut short in the mutiny, told me that, in a few years, he had so many operations, chiefly at *Saharunpore*, that he was able to take home (I believe) upwards of 300 calculi to his former teacher, Dr. Sharpey. Dr. A. Garden has reported³ 831 cases of stone treated in the Saharunpore dispensary in the 18 years ending July 1866. These cases probably include some of Mr. Heathcote's and Dr. Cockburn's noticed above. The late Dr. C. Manners Smith, of *Lahore*, operated in about 200 cases. His successor, Brigade-Surgeon John Barclay Scriven, had about 250 cases of lithotomy and lithotrity. Dr. John Ince has reported that 263 cases were operated upon in the *Dhera Ghazee Khan* dispensary in 13 years. Then, taking a southward line, Dr. De Renzy reported 352 operations from *Mooltan*. Much further south, Mr. Sub-Assistant-Surgeon Raddock performed lithotomy in 77 cases, men, women, and children, in about four years, while he was attached to the *Malwa Bheel Corps*. At *Goojerat*, Mr. G. E. Poole cut 85 persons for stone in 27 months. Dr. Keegan, Residency Surgeon, *Indore*, has recently published three reports⁴ of 208 cases of litholapaxy. Passing from Indore to the south and west, we have Dr. W. Burns Beatson's stone statistics of *Nagpoor* and *Raipur* which are given above—say, at the former, 12, and at the latter, which is situated 160 miles to the eastward, upwards of 200 annually.

URINARY CALCULUS—EUROPEAN TROOPS ADMITTED.

Year	Bengal	Madras	Bombay
1880	1	0	0
1881	0	0	0
1882	6	0	0
1883	1	0	0

¹ Most of the stones removed by this surgeon are preserved in glazed cases at the Calcutta Medical College Museum. The Bahadur has published a valuable monograph on his cases.

² *I. A.*, No. 23, p. 125.

³ *I. A.*, No. 23, p. 1.

⁴ *I. M. G.* up to September 1885.

NATIVE TROOPS.

Year	Regular Army of Bengal	Punjaub Frontier Force	Central India Force	Hyderabad Contingent	Army of Bombay	Army of Madras
1880	9	7, 1 died	1 died	2	1	4
1881	4	3	2	1	5	4
1882	6	3	1	2	5	0
1883	11	8	1	3	3	4

PRISONERS.

Year	Bengal Presidency	Andamans and Nicobars	Berars	British Burmah	Bombay Presidency	Madras Presidency
1880	10	0	0	1 died	1	1
1881	8	0	0	0	1	0
1882	6	0	0	0	0	0
1883	10	3	0	0	0	0

CAUSES OF STONE IN INDIA.

The above data appear to demonstrate the fact that circumstances of *Locality*, whether this may be the temperature, the atmospheric condition, the quality and the quantity of the staple food, the nature of the water supply, the habits of the people, or what not, have great concern in determining the rarity or excessive prevalence of calculous disease in India. Thus the influence of the sea-breeze, the impregnation of the soil with salt, the presence of terai malaria, the operation of the causes which, whatever their nature may be, give rise to bronchocele along the slopes of the great northern mountain range, peculiarity in the solid constituents of the water of hill streams, are all points deserving investigation as bearing upon the causation of stone. We have some rather definite facts with regard to the influence of FOOD.

In India, although the rich and well-to-do are not absolutely exempt from calculous disease, it is generally far less frequent among the wealthy there than it is among those who are quite above want at home. This may probably be due to the almost entire freedom of natives from gout. All observers concur in noticing that, especially in the North-Western Provinces, the subjects of stone are, for the most part, squalidly poor, ill-lodged, miserably fed, scarcely protected by clothing from the trying cold of winter, and drinkers of very impure water. The fact that people who are well-fed in India enjoy large immunity from calculous disease, is strongly confirmed in the above tables of native soldiers and prisoners. The greater tendency, however, to the occurrence of stone in certain districts in the northern

parts of the Bengal Presidency, especially below the slope of the Hinlaya, is distinctly shown in the tables, in regard to European soldiers, native troops, and prisoners. That, *in India, stone is common where the people are wheat-eaters, and unusual in the rice districts*, is a long-established and still unshaken fact.

Mr. Plowright, surgeon to the Norfolk and East Lynn Hospital, which receives a great many stone cases, has lately published¹ a very thoughtful and suggestive paper which merits the attention of the Indian Government and of all surgeons in our Eastern possessions. He shows that, although stone is notoriously very prevalent in Norfolk generally, there is a considerable district in which this disease is exceedingly infrequent. In its leading geological conditions, this expanse much resembles the Gangetic Delta. It is a tract of alluvial country of a triangular shape, bounded on the east, west, and north by the rivers Ouse and Nene, and by the Wash. It has an area of about 160 square miles. Its population is about 16,000. It has been reclaimed from the sea almost within historic times. Like the Delta of the Ganges, its soil is so impregnated with salt that only a few wells yield potable waters. Analysing the statistics of two agricultural populations nearly equal in numbers—that of Marshland and that of a corresponding district on the east side of the Ouse—he finds that some 20 cases have occurred in Marshland against 100 in the eastern area. Within the last twenty years, the proportions of cases have been: west of the Ouse (Marshland), population 16,634, 5 cases; east of the Ouse, population 17,825, 21 cases. He gives many statistical data which tend to show that, in various parts of England, stone had its greatest prevalence when the salt-tax was in operation. He cites Coupland Hutchinson, and other authorities, who have shown that calculous disease was exceedingly rare in the navy when oversalted provisions were in use; and argues that the great tendency to stone in the children of our poor arises from deficiency of salt in their food. Here I may mention that, up country, officers' children are not exempt from stone. I shall cite cases in which surgeons of my acquaintance cut their own children for stone. I never saw or heard of a case in a European gentleman's child in Lower Bengal. There, one of the commonest acts of a child is to take a handful—as often two—of salt from the salt-cellar, and to eat as much as it is allowed to do. I never checked this instinct. Mr. Plowright gives data for the conclusions that the presence of salt greatly increases the solubility of uric acid; that the consumption of salt, by increasing thirst, ensures a larger amount of fluid passing through the urinary tract, and therefore lessens the probability of calculus; and that, by keeping the colloids equally diffused, salt tends to prevent the crystalline solids of the urine

¹ 'On the Cause and Distribution of Calculous Disease,' *M. T.* vol. ii. for 1885.

from agglomerating into calculi. Lastly, he recommends a more liberal consumption of salt by those who are threatened with calculous disease. He urges that 'the disease is common in India, where salt is taxed.' Mr. Plowright's argument has been so ably stated that it recommends itself to the attention of all thoughtful and influential persons in the stone-districts of India.

We have seen that, throughout Eastern Bengal—taking the seaport of Chittagong, a great centre of the salt manufacture (the salt agent received, in my time, a salary of 2,400*l.* a year) as a starting-point, the late Kingdom of Burmah on one side, Assam to the north and the Gangetic Delta to the west, the whole of the Madras Presidency and the Presidency of Bombay—calculous disease is nowhere very prevalent. There, the sea-breeze generally extends some 250 miles inland, and salt, although not very cheap anywhere, is procurable by the poor.

From the foregoing remarks, it will be gathered that those districts of India in which calculous disease is prevalent are situated far inland, and are quite beyond range of the sea-breeze.

The whole of the Gangetic Delta, in a line west to east away from Burdwan to Dacca, is a salt alluvium, where the wells are brackish and the sand is so saline as to be unfit for building purposes. Here, as we have seen, calculous disease is rare.

At Cuttack, the capital of the great province of Orissa, where rice is the staple grain, stone occurs so very rarely that, in 1873, my friend Dr. Cayley had to remove three stones from the bladder of a very aged man without lithotomy instruments. Cuttack stands fully within range of the sea-breeze, not very distant from the vast Chilka Salt Lake, which is about 35 long by 8 miles broad.

That excellent observer, Dr. John Craig, has lately written to me : 'At Aden, the water used by the natives is brackish. I was there upwards of a year as medical superintendent, and never heard of a case of stone.'

It is very desirable, from a sanitary point of view, to ascertain whether the salt supply of the poor in the stone districts of Upper and Central India is adequate to their wants. This question is met by the difficulty that the masses of India are not only extremely poor, but are likewise (very unlike the labouring class in England), for the most part, abstinent to a degree, not only in regard to those articles which season food, but also in regard to the quantity of the food itself. It is therefore unsafe to declare that, in any Indian locality, the bazar supply of salt is so abundant and so cheap, as to bring the article liberally within the means of the poorest consumer.¹ 'The stone districts' are, for the most part, practically, as far removed from the supply of sea-salt as they are from the access of the sea-breeze. I am

¹ Many years ago I was told by a poor Welshwoman that, in her village, a large salmon might be got for sixpence—'but,' she added, 'nobody there has sixpence.'

mainly indebted for the following data to my friends Colonel Sibley, Surgeon-General Francis, and Dr. R. Pringle. The North-Western Provinces obtain their salt supply mainly (1) from the Sambre Salt Lake, in Ajmeer, west of Agra, which is about 13 miles long by two broad; (2) from the mines near Jhelum; (3) considerable quantities of rock salt are brought through the passes of the Himalayas, but this is a very precarious supply, and depends upon the passes being open and other circumstances. In the Punjaub, rock salt is almost exclusively used. The mining and preparation of salt is in the hands of Government, who sell it at fixed rates. The salt which is imported to our provinces from native States pays a duty; and there is (or was) a customs line all along our frontier. The restrictive influence of a salt duty is illustrated by the facts that my friend Mr. Joseph Bedford gave, in 1852, four rupees as the bazar rate of salt per maund (80 pounds) at Rampoor Bauleah, and that I learn, from indisputable authority, that the price of salt, up country, is, at present, two rupees or one rupee 14 annas per maund. I know, from having to diet Punjaubees in Calcutta, that these people flavour their thick 'elephant ear' chup-patties with lime, a national custom which probably took its rise from scarcity or dearth of salt. Writing of the N. W. Provinces, a brother officer says, 'The supply of salt is sufficient for man, but, as the value of hides increases, the supply available for men decreases, and I think cattle at all times suffer from its scarcity.'

A hint of the difficulty which, even in these days of railway communication, attends the supply of salt to remote inland districts, was given when Surgeon-General Francis wrote to me the other day, in reply to my inquiry whence the vast province of Bundelkund obtains its salt, 'I should not like to speak positively, but my impression is that it was *Lahoree neemuk*.'

It is an open question, but it is my distinct belief that the poor in the stone districts do not get enough salt.

Those who may wish to enter fully into the question of *the comparative success attending Lithotomy in India and at home* will find much valuable information in the subjoined memoirs.¹

The following is an abstract of a few of the results, as given by Greenhow.

Home.—Mr. Coulson found the death-rate in 1,743 cases to be one in 6·98, Sir H. Thompson in 1,827 cases to be one in 7·50, Fayrer, in France, 1 in 5·7; in Europe generally 1 in 5·14. Mr. Greenhow, after deducting 133 doubtful cases from 1,851 cases of lithotomy performed in the *North-Western Provinces*, finds that the death-rate, in

¹ Fayrer, as cited above; R. N. Dass, *op. cit.*; A. Simpson, *I. A.*, No. 3, p. 45; Durant, *Ibid.* No. 13, p. 78; Greenhow, *Ibid.* No. 22, p. 1; A. Garden, *Ibid.* No. 23, p. 1; Cutcliffe, at p. 124 of the same number; Poole, *I. M. G.*, vol. iii. p. 153; Cutcliffe, *Ibid.* vol. v. p. 153.

the remaining 1,718 cases, was 1 in 6·63. In 108 cases operated upon in *Lower Bengal*, (too small a number to draw a sound inference from) I find the death-rate to have been 1 in 4·8.

Greenhow gives the amount of success in *India*, as ranging from 1 death in 3 to 1 in 40.

In the chapter quoted above, Sir Joseph Fayrer gives an analysis of the causes of failure in Indian stone operations. Among these may be especially taken into account—malarious and scorbutic cachexia, a hospital abounding in the causes of hospitalism, a dispensary or a surgeon not yet famous for the treatment of stone cases:—in this case, patients often will not seek relief until their health is hopelessly broken. Surgeons used to say that, unless you operate upon native patients immediately upon admission, they will not remain. When on tour with the Governor-General, Mr. Brett would operate upon a patient under a tree, and leave him to the care of his friends. 'It was said of the late Dr. A. Simpson that he would get out of his palanquin and relieve, in the same manner, patients who came up imploring his assistance. Often a man will be removed by his friends before the wound is healed. Clearly there must be an undue rate of mortality among such unprepared and impatient sufferers.

In his long and very extensive experience as a lithotomist in the N. W. Provinces, my friend Surgeon-General Cockburn, I think very wisely, operated upon every native who came to him with stone, whatever the concurrent circumstances might be, the necessity of relieving the poor creature's suffering standing before every other consideration.

He tells me that he learnt afterwards that an old native man bathed in a tank every morning after the day upon which he was cut, and yet made a good recovery.

Fayrer gleaned from the records of the Medical Department that the death-rate in 554 cases of lithotomy performed in six months of 1863, in the Punjaub, N. W. and Central Provinces, was nearly 1 in 10. Hence I think we may infer that, in an Indian climate where paludal cachexia does not abound, and at dispensaries in which sufferers are anxious to seek relief early, a very large measure of success may be anticipated.

I have noticed, elsewhere in this work, that one of our chief disappointments and causes of ill-success in treating natives, arises from the tendency of the patients and their friends to turn their backs upon us just as their cases are becoming interesting or critical. An observation by Dr. G. Henderson, however, shows that even this great evil may sometimes be turned to good. He says,¹ 'In some cases' [of lithotomy] 'a week or so after operation, diarrhœa comes on, or the patient gets obstinate fever or some other complication, and begins

¹ *Notes on Lithotomy*, I. M. G., vol. ii. p. 40.

steadily to go down-hill in spite of treatment. In such cases, ought we to trust to treatment in hospital, or yield to the urgent request of the patient and his relatives to allow him to be carried to his home? At first, I was strongly opposed to this practice; but, some years ago, a moribund patient was carried off by his relatives during the night without my consent, and he was entered in the register as dead; but, six months afterwards, he appeared to thank me for having relieved him of the stone. Since that time, I have made it a rule that, as soon as I see no reasonable hope of recovery after the operation of lithotomy, and after all resources have failed, to suggest to the relatives that he should be carried home, provided he lives within twenty miles and wishes to be removed. Such a case I enter in my notes as fatal, unless I afterwards get evidence to the contrary. The effect of change is quite wonderful in most cases. Only the other day, a strong healthy man who was operated upon by me got diarrhœa on the eighth day; all remedies failed to check it, he began to despond, and I suggested that he should be taken home, a distance of five miles, and I gave him some astringents to take with him; four days afterwards, his wife returned to tell me that, from the time he left the hospital, he began to improve, and was then quite well. It is certain that the air of a crowded hospital, in the heart of a populous native city, is unfavourable for recovery. The plan adopted by me will probably be opposed by many surgeons; but, in a certain proportion of cases—and the number is very small—I feel certain that it will save lives that would otherwise be lost. The same good effect is sometimes seen when a moribund prisoner is released from jail and made over to his relatives to be carried to his home; the effect is magical.' (See remarks on Removal, p. 173, and on Ichoræmic Diarrhœa, p. 577.)

Composition of Calculi.

I do not find any considerable series of analyses of calculi removed from *Bengalis*. This is a desideratum. Fayrer gives two cases: a Bengali gentleman, uric acid coated with triple phosphates, and a Bengali police officer, mulberry calculus coated with phosphates.

In a report of eleven stone cases upon which he operated at *Dacca*, the late Mr. Cutcliffe speaks of 'an oxalate of lime calculus,' 'a phosphatic stone,' 'a lithic acid stone,' 'a stone apparently lithic acid,' and 'a stone apparently phosphatic;' but he does not give any analyses.

Analyses of many calculi removed *Up-Country* are given, especially by Simpson, Durant, Greenhow, Garden, and Cutcliffe.¹ Mr. Durant's analysis of 21 stones removed at Mooteeharee will be found at p. 78, No. 13, of the 'I. A.' The main results are, triple phosphate 66·6 per cent., oxalic 19·0, and lithic 14·2. The analyses given by Simpson,

¹ As cited above.

Greenhow, and Garden, show that the deposits prevailed in the following proportions: urates, 333; phosphates, 193; oxalates, 87.

In the work cited above, Ram Narain Das, Bahadur, gives beautifully executed lithographs of 127 calculi, with careful analyses in which *the composition of the Nucleus* is particularised. In 59, the nucleus was chiefly of uric acid, with occasionally admixture of urate of ammonia and of oxalate of lime. In 53, it was either wholly or principally of urate of ammonia. In 8, it was composed in part or wholly of oxalate of lime.

In the Catalogue of the Grant M. C. M., Bombay (*vide* remarks at p. 675 on calculous disease in Bombay) is a report by Dr. H. Vandyke Carter upon the analysis of the urinary calculi, in which *the composition of the Nuclei* is carefully specified.

CALCULI ARRANGED ACCORDING TO NUCLEI.

Series I.—Uric Acid.

	Number
Uric Acid	4
„ Urate of Ammonia	6
„ Earthy Phosphates	2
„ Urate of Ammonia, Uric Acid	1
„ Oxalate of Lime, Earthy Phosphates	1

Series II.—Urate of Ammonia.

Urate of Ammonia	6
„ Uric Acid	2
„ Oxalate of Lime	19
„ Earthy Phosphates	9
„ Uric Acid, Urate of Ammonia	1
„ „ Oxalate of Lime	1
„ „ Earthy Phosphates	1
„ Oxalate of Lime, Uric Acid	3
„ „ Urate of Ammonia	2
„ „ Earthy Phosphates	8
„ succeeded by 4 or more layers	1

Series III.—Oxalate of Lime.

Oxalate of Lime	16
„ Uric Acid	2
„ Urate of Ammonia	2
„ Earthy Phosphates	10
„ Uric Acid, Oxalate of Lime	1
„ Urate of Ammonia, Uric Acid	3
„ „ Oxalate of Lime	1
„ „ Earthy Phosphates	3
„ succeeded by 4 or more layers	1
„ Phosphate of Lime, Oxalate of Lime	4

Series IV. to X.

Cystic Oxide	1
Triple Phosphate	1
Fusible Calculus	1
Phosphate of Lime	2
Animal matter	1

India appears always to have supported hakeems and others of a lower grade, practising systematically as lithotomists, who operate 'on the grip.'¹

Mr. J. H. Hindley has given, in the 8th vol. of the 'I. M. G.,' p. 39, an account of stone operations performed in Marwar by a cultivator, an inhabitant of the hilly tracts of Sirohi, as well as representations of the knife and hook which he used. Dr. R. Temple Wright has also published, in vol. xi. p. 1 of the same periodical, representations of native barbers' common razors (*ustārā*) used by *Jurrars*, with the curved forceps (*zūmboōr*), in lithotomising. Dr. Wright says that native surgeons in the North of India, the Punjaub, and N. W. Provinces, although they have had no professional education, perform lithotomy with great success—96 per cent. of recoveries being a common rate. They are, doubtless, careful to pick their cases. Mr. Hindley remarks that, in Marwar, 'only strong healthy persons, in the prime of life, were treated' by the operators. In some of the survivors, the perineal wound remains fistulous.

The following singular case² appears to show that there is yet a lower depth even in the practice of these native stone-cutters. A boy, about ten years old, was brought to Mr. John Morice, at the Gwalior Dispensary, with symptoms of stone and with an opening in the left groin through which the urine dribbled. The father said that, about four years previously, a 'hakeem' in the city had operated, making the wound in the groin and removing a stone or part of one. The wound remaining open and symptoms of stone continuing, he took the boy to Agra, where a 'medical officer' re-opened the wound in the groin and removed a stone or part of one. During the subsequent two years, he had passed his urine through the wound. A large stone, of a curved shape, with one end adherent to the bladder, was removed by the lateral operation on the right side. It weighed 460 grains. In fifteen days, the urine all came by the urethra and, stimulants being applied, the old wound healed.

The triumphs of English lithotomists in India commenced within the memory of some Company's surgeons still living. Dr. Finch observed, in 1834,³ that it appeared that lithotomy had not, until very

¹ See descriptions of the native operation by Mr. G. Playfair and Mr. Brett, *I. M. J.*, vol. i. N. S., p. 429; by Dr. Percival Lord, *Transactions of the Medical and Physical Society of Bombay*, 1838; and by Mr. A. Gibson, *Ibid.* for 1840.

² *I. M. G.*, vol. i. p. 118.

³ *I. M. J.*, vol. i. p. 322.

lately, been undertaken in India by European surgeons. In April of that year, he operated upon a boy with 'a grooved staff made up by a *lohar mistree* (blacksmith) of the regiment, a scalpel, and ball forceps.'

The papers cited above are replete with valuable and striking facts well meriting the attention of all lithotomists. Did space allow, I could give many remarkable illustrations of the difficulties and the triumphs which have fallen to the lot of operators in the East. Mr. James McRae, one of my predecessors in the Principalship of the Medical College, a very eminent surgeon, being posted beyond the reach of other aid, operated successfully upon his own son, three years of age. The stone, of the size of a large nutmeg, weighed three drachms.¹ Mr. W. Bramlet Switzer also operated upon his own child. The little fellow was only eleven months old. He was dying of irritative fever, and his father's resolution saved his life. The stone, which was of the size of a garden bean, was removed by a dressing-forceps.

Dr. J. Wilson Johnston, civil surgeon of Loodiana, wrote to Dr. Greenhow: 'A native consulted me about his wife; he objected to my seeing her, but was determined to obtain relief. His description left no doubt in my mind as to what was the matter. Ultimately, I undertook to extract a calculus, without seeing or touching her, without drawing a drop of blood. She was placed behind a *pardah* (curtain), on a table, and directions were given for introducing a sound; this done, it was placed in my hand. I soon detected a calculus of no great dimensions. Weiss's dilator, closed, was now introduced as before; grasping it, I cautiously dilated and passed a small-sized forceps, and this concluded the affair successfully.'

I have heard of more than one case in which our surgeons have removed calculi left some time previously in the bladder by native lithotomists. I saw Sir Joseph Fayrer's 'thirteenth case.'² An Englishman, *æt.* 44, while working upon the side of an embankment, fell and slid down—a stick entered the rectum and wounded the bladder; consequently, there were a recto-vesical fistula and a calculus. The stone was removed by the lateral operation. He died of pyæmia.

Dr. Kenneth McLeod³ had a Mohammedan patient, *æt.* 27, who stated that, in descending from a roof of a hut which he was repairing, his foot slipped, and a pointed bamboo needle (*shamajee*), used needle-like in thatching, entered his perineum. There was no escape of urine. A small cicatrix appeared about an inch to the right of the anus. Upon finding that the bladder contained a foreign body, Dr. McLeod lithotomised and removed a pointed bit of bamboo $2\frac{3}{4}$ inches long, $\frac{1}{8}$ inch in diameter at the point, and $\frac{1}{4}$ inch in diameter at

¹ He reported the case in the *I. M. J.*, vol. ix. O. S., p. 275.

² *Observations*, p. 389.

³ *I. M. G.*, vol. iv. p. 141.

the base. It was partly crusted by deposit. The extremities were smooth.

Dr. J. E. B. Aitchison relates the case of a native, *æt.* 25, from whom a fakir attempted to remove a stone by cutting into the bladder by the right side of the perineum. He succeeded in getting into the bladder, but could not extract the stone, which broke at every grasp, small pieces coming away. At first, urine was passed by the perineal wound. Latterly, it came thence by drops. The patient was in a miserably low condition; worn away to a skeleton, suffering intense agony, and having a tendency to trismus. After four days' preparation, Dr. Aitchison operated on the right side. The tissues were more or less consolidated into one, and free incisions were now necessary, as the parts would not dilate as easily as in the normal condition. Two large, soft, and very friable stones were removed. It took fifteen minutes to remove the second, which had a large facet and was very peculiarly formed. As he was weak, he made a slow but perfect recovery.¹

I had in my ward a man who had twice undergone the lateral operation for stone in America. Assistant-Surgeon T. C. Bhadoory has recorded² the case of a Hindoo cultivator (*æt.* 25) in whom lithotomy had been performed four times, twice by the reporter. The operations (lateral) were through the cicatrix. In three of the operations, the stone broke during extraction, but the bladder was washed out with copious injections, and care was taken to examine thoroughly the interior of the viscus, to ascertain that no fragments were left behind. On the third occasion, the calculus was extracted entire. He considered that the recurrence was due to an obstinate continuance of the calculous diathesis.

Dr. Cayley successfully removed, by lithotomy, three stones from the bladder of a native believed to be ninety years of age, who was accompanied to the dispensary by his son, a grey-haired man of sixty.

I think that Indian medical men have never been tardy in making trial of every improvement in the *ars medendi* which has deservedly obtained reputation in Europe. In 1832, Dr. J. N. Casanova performed, in Calcutta, two *Lithotritry* operations with instruments which he had obtained from Civiale.³

In 1867, Mr. Greenhow gave a brief account of the small progress of lithotritry in India. He writes: 'The information regarding lithotritry is but small, few surgeons considering it an operation suited to the natives of India. Dr. Smith, of Lahore, and Mr. Lyons have tried it a few times, but both prefer lithotomy.' At the Lahore Medical College, there occurred, during the three years commencing 1861,

¹ *I. A.*, No. 5, p. 266.

² *I. M. G.* for 1872, p. 89.

³ *C. M. T.*, vol. vi. p. 435.

'fifteen cases of lithotrity, of which 8 were cured, one died, and 6 were uncertain in their result, principally because the patients left before recovery was complete. The Principal, Mr. Scriven, advocates lithotrity in old men and in women, and remarks, 'I consider it much preferable to lithotomy for women, for, though the latter operation can scarcely be said to be dangerous to life in the female, it is very often followed by incontinence of urine which renders the patient's life permanently miserable.'

Later on,¹ Mr. Scriven gave a brief account of thirty-three cases of lithotrity which he had treated since 1861 at the hospital of the Lahore Medical School; of these, 29 were males and 4 females. Of the males, 19 recovered, one of them having undergone lithotomy; 7 left the hospital of their own accord, 4 being relieved, and 3 no better. One only died in hospital. Of the females, lithectomy was performed in one. All recovered. Other lithotrity cases are given by Mr. Scriven, 'I. M. G.' for April 1869, p. 69.

Dr. P. J. Freyer has published² a report of seventy-seven cases of natives in whom *Litholapaxy*, or lithotrity at one sitting, was performed. All were successful save three fatal cases. This gentleman has subsequently published, in Calcutta, a monograph detailing the results of a hundred and eleven of these operations.

Up to September 1885,³ Dr. Keegan, Residency Surgeon of Indore, has reported 208 cases of litholapaxy in male children and adults, with 17 deaths.

Dr. W. Palmer has recorded⁴ some valuable observations on '*the remarkable power possessed by the alkaline hyposulphites in arresting the formation of pus in the urinary bladder*,' particularly the case of a man who had been paraplegic for years, who was nearly worn out from bed-sores, and the constant drain of from 6 to 10 ounces of pus from the bladder daily. Half a drachm of hyposulphite was given to him every three hours for six days, when the urine became clear and quite free from pus or putrescence. After four days, there was again as much pus in the urine as ever. Similar doses were again ordered, and the result was a total disappearance of pus after four days. Beyond this, the patient was too near death to be materially improved.

A man suffering from strictures passed from 4 to 6 ounces of pus from the bladder daily. Half-drachm doses of hyposulphite of soda were given every three hours, and the strictures were treated by gradual dilatation. The amount of pus in the urine decreased rapidly; in five days, it had entirely disappeared, though it was six weeks before the patient made a perfectly free stream of urine.

¹ *I. M. G.* for August and October 1868.

² *Ibid.* for December 1882 and February 1883.

³ *Ibid.* for that date.

⁴ *Ibid.*, vol. iv. p. 116.

Cases of *Prostatic Calculi* occur in India from time to time.

Urethral Calculi.—The lodgment of a calculus in the urethra is a frequent occurrence. In 107 cases of stone, Dr. P. J. Freyer had 6 of urethral calculus. Several Indian examples tend to demonstrate the fact that, in neglected cases, where a stone has been just too large to escape from its lodgment in the urethra, urine is passed, not unfrequently for years, and accretion goes on until the stone acquires a formidable size, as in the following cases :

Mr. J. W. Blackwell reported¹ the case of a Hindoo of Vizagapatam, *æt.* 22, who had suffered from difficulty in passing urine for 14 years. Latterly, the pain was most agonising and the calls most incessant. He said that every half-hour, day and night, he had to throw himself into every imaginable attitude, frequently standing on his head, to be enabled to pass a few drops ; the bladder was never properly emptied. He was emaciated and cachectic. A sound struck on a stone at the anterior part of the prostate gland, and the finger in the rectum detected a large bulging mass, quite immovable. A sound was passed to the stone, and an incision carried on to it. It was removed in two pieces ; to bring away the second portion it was necessary to divide the prostate, to which it appeared to be firmly adherent. The stone was of triple phosphate, and weighed $3\frac{1}{2}$ ounces. His bowels became disordered and he died on the fifteenth day.

Sub-A.-S. Mohim Chunder Roy² had a young patient in whom a stone, as large as a betel-nut, lay posterior to the scrotum. The urethra behind was enormously dilated and filled with stones. He removed, by an incision, this stone and about two ounces of small stones. These were reported by the sweeper to have been stolen—the people of Nowkhilla administering urinary calculi, ‘rubbed on iron,’ to patients suffering from stone.

A case of this kind occurred in the practice of Mr. Hussey.³ A child, 18 months old, was brought, in a moribund state, to the Radcliffe Infirmary, suffering from symptoms of stone in the bladder. On sounding, a calculus was felt with the curve of the staff, but so far forward that it seemed that the stone was not in the bladder. Upon examination *p. m.*, the bladder was contracted, and its coats thickened. A large soft calculus, composed of triple phosphates, a drachm and a half in weight, was found in a cyst in the membranous portion of the urethra.

In the four following cases, perineal abscess appears to have occurred. Baboo B. M. Bose treated a Hindoo boy, *æt.* 14, who had a stony tumour, about the size of a duck’s egg, lying between the testicles, and having a slender, hard, and gritty prolongation towards the root of the penis. There was constant dribbling of urine through a

¹ *I. A.*, No. 4, p. 701.

² *I. M. G.*, vol. viii.

³ *Miscellanea Medico-Chirurgica*, p. 119.

sinus communicating with the fossa navicularis. The external meatus only admitted a director with difficulty. The deposit was considered to have originated in a perineal abscess, the fistula of which had healed externally, four years previously. The stones were removed by an incision in the line of the urethra. The largest stone weighed nearly an ounce.¹

Dr. Cleghorn² had a Hindoo patient, *æt.* 55, who, 15 years previously, suddenly experienced great difficulty in passing urine. After a time, this difficulty diminished, but the act was always accompanied with a good deal of straining. Six years later, perineal abscesses formed and discharged a small stone. Through these, the urine passed. A catheter struck a stone in the urethra anterior to the scrotum, but could, with difficulty, be passed into the bladder. The stone was removed by an incision which divided the fistulous openings. The phosphatic stone is described as resembling an enlarged prostate in shape, and as being of the size of a large egg. It 'was divided into two lobes by a deep groove, caused by the current of urine passing over it. It weighed 2 oz. 7 drs. 10 grs.'

In an Afghan, *æt.* 50, who was stated to have been operated upon for stone by a hakeem some years previously, fifty calculi, of various sizes from that of an almond shell to that of a small pea, were extracted from two cysts, in perineo, which communicated freely with bladder and urethra. A staff could only be introduced as far as the calculi, which could be distinctly felt with the fingers externally, but it was not considered that there had been any perineal fistula. The stones were so firmly adherent to their sacs that much difficulty was experienced in removing them. He was discharged cured in a few weeks.³

In sounding a Hindoo labourer of the Dacca district, *æt.* 50, who had suffered for two years from symptoms of stone, the late Mr. Cutcliffe⁴ ascertained that there were many calculi in the bladder, and a peculiar fulness and hardness in the perineum. It was found that a nest of calculi occupied the recto-vesical space, being contained in a pouch quite large enough to be mistaken for the bladder itself. Within the bladder were as many calculi as were contained in the perineal sac. These latter were considered to have found their way out by ulceration of the neck of the bladder. Altogether, there was a large handful of phosphatic calculi, weighing an ounce and a half and twenty grains. The patient had a good recovery.

One desiring to write a book upon the *vires medicatrices Naturæ* would probably value the following case, by Mr. J. O'D. Partridge,⁵ as showing how effectually a calculus may find its own way out of the bladder. A native boy, *æt.* 7, had inflammation of the left scrotum

¹ *I. M. G.*, vol. viii. p. 15.

² *I. M. G.*, vol. ii. p. 103.

³ Webb's *Pathologia Indica*, p. 240.

⁴ *I. M. G.*, vol. v. p. 81.

⁵ *Ibid.* vol. vi. p. 36.

and [perineum?]. He made water freely. On the following day, a black spot (sloughing) appeared on the scrotum. On the sixth day, the scrotal ulcer was completely open; and, through it, a calculus, weighing 100 grains, passed, and was followed by a discharge of urine. There was also an abscess on the opposite side. The history is only pursued up to the next day, when the patient was 'improving and the wound healthy.' The father, who, I suspect, was capable of overlooking a great deal, stated that the child complained of pain four days before, but had never been ill previously, shown any sign of pain, or had any difficulty in passing his urine.

Surgeon-General W. J. Moore speaks¹ of a case in which 'a stone passed by abscess and ulceration from the prostatic portion of the urethra, or from the base of the bladder, through the perineum.'

These cases demonstrate the law that vesical calculus, when left to itself, has a tendency to make its way out through the perineum.

Prep. 885, in the C. M. C. M., is 'a calculus as large as a small hazel-nut impacted behind the prepuce. It is *in situ*, and the prepuce is seen much tumefied. The stone has escaped from the urethra, and now appears underneath the integument.'

Mr. James Boyd, civil surgeon of Tannah,² treated a villager who, about a year and a half previously, first perceived a small round body near the extremity of the urethra. At first, it caused no inconvenience, but, as it gradually increased in size, the stream of urine became diminished. He was now suffering from retention of urine occasioned by a calculus, producing considerable enlargement, impacted near the mouth of the urethra. It was removed by incision. It weighed nineteen grains, was made up of concentric lamellæ, and was composed of carbonate of lime.

In the case of an Oorya, who had not made water easily for some months and in whom retention had been present for two days, Allan Webb found a calculus lodged at the root of the penis. He brought it down, as far as the glans, with a bent probe and freed it by an incision.³

My friend Dr. Herbert Baillie pointed out⁴ that the dislodgment of a calculus impacted in the urethra is much facilitated by passing down to it a director the groove of which is charged with oil; the instrument is then moved about, so as not only to dilate the canal, but likewise lubricate it, so that a little manipulation forces the stone forward to the meatus, or at least to a little behind it, whence it can be readily extricated with a scoop.

Preputial Calculi now and then complicate phimosis.

Dr. J. Macpherson showed, at a meeting of the Medical and Phy-

¹ 'Native Practice in Rajpootana,' *I. A.*, No. 21, p. 106.

² *I. M. G.*, N. S., vol. i. p. 680.

³ *Pathologia Indica*, p. 233.

⁴ *I. M. G.*, vol. v. p. 36.

sical Society of Calcutta, a calculus, of the size of an ordinary nutmeg which he had removed from the prepuce.

Mr. L. F. Dickson mentions¹ the case of a Hindoo with congenital phimosis, *æt.* 45, from whom 52 preputial calculi, from the size of a mustard-seed to that of a pea, were removed by circumcision.

Sub-Assistant-Surgeon Samachurn Dutt² operated on a case of phimosis in which about 200 calculi had formed. The orifice of the prepuce was almost entirely closed, so that the urine was passed with difficulty.

In 1867, the curator of the Calcutta Medical College Museum received, from Dr. D. Wright, of Khatmandoo, 'a collection of urinary calculi removed from the prepuce of a middle-aged man by N. D. Narayan Dobey.'

In a Brahmin, *æt.* 28, Dr. R. G. Matthew³ found, at the end of the penis, a spherical tumour about the size of a French billiard ball. Its surface was traversed by large tortuous veins. The preputial orifice was extremely small and was situated below and posteriorly. On dividing the enormously thickened tissues, a stone, about seven inches in circumference, and weighing seven ounces and three drachms, was removed. After circumcision, the man made a good recovery. There was a cup-shaped depression on the stone in which the glans (which it was thought was seriously damaged) was lodged. The disease was of twelve years' standing. It was added that a case had occurred to Dr. Wishaw in which 420 stones, varying in size from a bean to a pin's head, were found in the prepuce of a native patient.

The following odd case by Dr. Crombie⁴ belongs equally to medical jurisprudence and to surgery. A young Hindoo of Dacca came to the dispensary saying that he had introduced a wire into his urethra. A foreign body was detected in the membranous portion, was cut down upon and removed, but it did not come away at all readily. It proved to be a piece of thorny '*bent*' about five inches long. It had 18 thorns or hooks on it in sets of three, placed alternately on opposite sides of the *bent*. Two or three of these had been broken off in dragging it out; he, however, made a satisfactory recovery in three weeks. It had been introduced by the husband and friends of a woman with whom he had been over-intimate.

I have noted, in my 'Medical Jurisprudence,' p. 556, that the 'introduction of a straw or wire into the urethra' is mentioned, in the Madras report on torture, as a cruelty practised in that Presidency. I have not heard of another case in Bengal.

Assistant-Surgeon Khazan Chand has published⁵ an almost unique case of hæmaturia due to the presence of a *Leech in the Urethra* of a

¹ *I. M. G.*, vol. viii, p. 124.

² Webb's *Pathologia Indica*, p. 235.

³ *I. M. G.*, March 1886.

⁴ *Ibid.*, September 1878.

⁵ *Ibid.*, January 1885.

Mohammedan male, *æt.* 48. The bleeding from the urethra was enormous, and the patient was mentally depressed. Injections of alum and nitrate of silver were used. On the third day, bleeding was still abundant. The head of a leech protruded and was drawn out; bleeding then ceased. He said that he used to bathe in a tank where leeches were abundant.

The preparation numbered 854 in Ewart's Catalogue displays a *fistula leading from the bladder through the abdominal parietes*, 'and communicating' [opening?] 'externally just underneath the umbilicus, from a paraplegic native woman. The bladder is small, with the mucous membrane of the fundus ulcerated.'

This case illustrates a natural law under which the bladder, when over-distended, is liable to give way in the direction of the urachus. The six cases which are subjoined appear to confirm this fact.

Mr. J. Davidson placed on record¹ the case of a stout middle-aged Mohammedan suffering from retention of urine which he stated came on gradually, but no urine had been passed for two days. The bladder was painfully distended, and was felt, very large and hard, above the pubes, and the penis and scrotum were oedematously swollen. A very large quantity of very fœtid urine was easily drawn off. Great relief followed, and he continued to pass urine pretty copiously, but with gradually increasing pain and difficulty. This seemed attributable to a swelling, just above the pubes, which was as large as a goose-egg, hard and puffy; the swelling had extended to the penis, and the urethra appeared obstructed by it, so that he could not pass urine for several hours. A free incision gave vent to about eight ounces of fœtid pus and sloughs. In about half an hour, urine was passed by the urethra; but, during the night, it also escaped by the wound. The discharge of sloughs and urine mixed with pus continued for upwards of a month, during the early part of which he suffered from fever and bowel-complaint; but, eventually, the abscess healed and he was restored to good health.

Dr. R. H. Stevens reported² the case of a Hindoo, *æt.* 16, suffering from an abscess in the hypogastrium simulating a distended bladder. There was inability to pass urine, and great pain in the effort. There had been fever and pain in urinating for six days. The bladder had not been relieved for 24 hours. A number six catheter was passed, only four ounces of urine were drawn off, and pain and distress were not relieved. Paracentesis with a small trocar emptied the abscess. Recovery was speedy. Mr. F. M. Mackenzie³ had a very similar case in a Hindoo *æt.* 20. On *p.-m.* examination, an abscess was found, containing pus and blood, between the bladder and the abdominal wall. No cause could be assigned.

¹ *C. M. T.*, vol. vii. part i. p. 16.

² *I. M. G.*, vol. vii. p. 274.

³ *Ibid.* p. 208.

Dr. Kenneth Mackinnon related¹ the case of a man who was brought to him, having been subject for ten days to retention of urine. He was in a state of collapse. On passing a catheter, no urine flowed. The lower part of the belly being very tense and painful, with fluctuation, a trocar was plunged in about an inch below the umbilicus. Not less than four quarts of thick bloody urine came away. The man was relieved, but sank. No *p.-m.* was allowed. It seems evident that the bladder had given way where it is covered by the peritoneum.

A case in which the bladder gave way at its fundus is described by Mr. Hussey.² An unhealthy labouring man, *æt.* 48, sank while under treatment for retention of urine. Some days before death, a swelling was observed in the umbilical region, a little to one side of the median line. It was tender on pressure; the skin over it was slightly reddened, and, in general appearance, it was like a collection of matter in course of formation in the parietes. This subsided. Upon opening the abdomen after death, there was an escape of about a pint of thick and foul urine. It was contained in a sac formed in front by the parietes and behind by the mass of intestines, which were matted together with lymph. This sac was lined throughout with a thick layer of false membrane, not unlike the cavity of an abscess. A round opening, large enough to admit the finger, communicated with the fundus of the bladder. The bladder was contracted, and had, on its inner surface, some nodules which appeared to contain cancer-cells.

Mr. McReddie punctured the bladder above the pubes in a native who had not passed urine for eight days, and who was moribund with perineal extravasation and a sphacelated scrotum. The anterior wall of the bladder which, when distended, reached the umbilicus, 'had contracted adhesions with the abdominal parietes, and seemed to be in course of forming an abscess for evacuating urine, thus attempting what the operation performed.'³

My friend Dr. John Macpherson has related⁴ the very remarkable case of an Englishman, *æt.* 31, who had suffered from hæmoptysis and who had gradually increasing difficulty in passing water, although there was no impediment to the introduction of a catheter. He was of phthisical aspect and had pain and swelling in his left groin, where several small glands were enlarged. The thigh became very painful; he kept the knee constantly flexed, and he could not stand. About a month later, there was difficulty in making water. A full-sized catheter passed readily; the urine deposited an immense purulent sediment. In the three following months, the urine was always loaded with pus;

¹ *I. M. J.*, vol. iii. N. S., p. 274.

² *Miscellanea Medico-Chirurgica*, p. 150.

³ *I. M. G.*, vol. i. p. 5.

⁴ *I. A.*, No. 2, p. 586.

there was troublesome cough with copious expectoration. He gradually lost strength, so that a fatal result was prognosticated. There was constant pain in the loins. In the fourth month of observation, there was increased swelling in the thigh, and it began to be localised over the trochanter, his mouth got aphthous, the cough again became more troublesome, the urine was still purulent, and there was some pain in the loins. A month later, an opening was made in the swelling over the trochanter, and there was a copious discharge of healthy matter. A probe could be introduced nearly its whole length in almost every direction from the opening, but no bone was exposed. From that moment, the urine became clear. On the following day, he declared that urine had passed from the opening, and Dr. Macpherson was convinced by seeing it flow in a stream. Fully one half of his urine escaped through this novel channel. In the seventh month, he was improving in general health, the urine had almost ceased to flow through the fistula, but there was more swelling about the hip, and the left foot was swollen and œdematous. At the beginning of the eighth month, it was reported that the fistula over the trochanter had been closed for a fortnight, urine clear, cough gone, and he was gradually regaining his strength. Two years and six months subsequently, he wrote that his constitution still remained firm; he often rode twenty-four miles a day. No cough, water clear and free, particularly during the last twelvemonth. No pain in the loins, neither swelling nor uneasiness in the seat of the abscess; slight transitory stiffness in the left extremity.

I think it possible that, here, a chronic iliac abscess, of strumous origin, opened first into the bladder and afterwards through the bone.

Although there are many recorded cases of *Recovery from Accidental Wounds of the Bladder*, all additional instances are valuable. The following may be ranked with the cases by Sir Joseph Fayrer and Dr. Kenneth McLeod in which sharpened sticks entered the bladder by the perineum or rectum and broke in the cavity (p. 686). Dr. J. A. Greene¹ treated a Mohammedan boy, *æt.* 12, who, three days before he was brought in, was knocked down and gored by a cow. He had a lacerated wound two inches long, half an inch above and parallel to Poupart's ligament on the left side; the lips of this wound were sloughy and half an inch apart. Through this aperture, urine was constantly flowing, sometimes with a gush. The separated lips of the wounded bladder had formed adhesions to the parietes of the abdomen, but some urine had escaped into the peritoneal cavity. There was peritonitis, the abdominal muscles were as hard as boards, there were great pain and tenderness at the least pressure all over the abdomen; the body was bent forwards, and the knees drawn up. The bowels had not

¹ *I. M. G.*, vol. vi. p. 140.

been moved since the accident, nor had any urine been passed by the urethra. Half a grain of opium was given every three hours. A piece of lint was kept constantly on the wound, and warm fomentations were applied to the abdomen. Milk diet. His bowels were moved on the fourth day after admission, and he passed urine by the natural passage. The wound gradually closed, and the peritonitis subsided. He left well on the twelfth day after admission. Dr. Greene judiciously refrained from using the catheter.

Senile Disease of the Prostate—Early in his career, Mr. J. F. France wrote that the food of one threatened with prostatic enlargement ought to resemble, as much as possible, that of a graminivorous animal. Prostatic disease is very rare among the grain-eating natives of India. Ewart's Catalogue of the Calcutta M. C. Museum does not indicate a specimen. The Bombay and Madras collections each contain one specimen (marked, respectively, F. 18, and 27 Series 11), but the races of the individuals are not noted. I recollect a case which was more amusing than instructive. On joining a new station early in my Indian service, I received a message to the effect that one of my native assistants, who had charge of an important branch dispensary seven miles off, was bringing to me an old native man, with retention of urine, in whom every attempt to relieve the bladder had failed. I had been told that the Baboo was a very distinguished gold-medallist and scholarship holder, and it must be confessed that, in secret, I was rather inclined to feel in awe of him. On their arrival at my dispensary, I had everything set in order with a view to opening the bladder, and discussed with my learned friend an operation which had, just then, been strongly recommended. I then said that, of course, merely as a matter of routine, every attempt at catheterism having already failed, we would place the patient in a warm bath and use the catheter. Taking my largest and heaviest prostate catheter, I passed it with the greatest facility. Upon this, my friend and I looked at each other in speechless astonishment, and then his eye followed mine to the great armamentarium of surgical instruments set forth upon a side table. My really rather accomplished friend had never learnt to pass a catheter; and, for some time, I had to travel 14 miles daily until the patient was able to urinate.

Under the head of *Hæmorrhoids*, I have mentioned (p. 588) a case of long-continued retention of urine caused by the pressure of a hæmorrhoidal tumour. In a young native man who fell and struck his perineum on a narrow beam along which he was walking, and in an aged native, upon whom the thatch of a hut fell, there was *Extravasation of Urine*. I passed a female catheter through a free perineal incision and kept it in the bladder until the sloughs came away. Both made complete recoveries, although there were two attacks of copious venous hæmorrhage, controlled by plugging, in the elder man. I have spoken of *Extravasa-*

tion of Urine in cases of Perineal Abscess, under the head of Malarial Cachexia, p. 256.

Stricture of the Urethra.—I wrote, early in my Indian service, that this condition is rarely seen in natives of Lower Bengal. Ewart's Catalogue of our Museum contains mention of only three specimens. I infer that the subject of No. 862—'an impervious stricture of the membranous portion of the urethra'—was a native, as the specimen came from the dissecting-room.

In the native army of India, the strength of which averaged 114,612, there were, in 1881, 987 admissions for gonorrhœa. The admissions for stricture were only 39, with one death; whereas, in the European army, averaging 58,728 strong, the number invalided for stricture of the urethra was 42 out of 266 treated. 88 prisoners were admitted. It is probable that a great many native soldiers, suffering from gonorrhœa and stricture, contrive to obtain treatment outside the hospitals. Consequently, the admission-rate may inadequately represent the prevalence of these diseases. Still the above statistics show that, when the native soldier suffers very greatly from the disease, he is apt to seek hospital relief. Hence it must, I think, be inferred that severe stricture is rare among these men.

Out of 42 cases of stricture, treated at the Calcutta M. C. H., reported in Fayrer's 'Observations,' 12 were in Hindoos, 7 in Mohammedans, 7 in East Indians, and 16 in Europeans.

In a paper on 'Stricture of the Urethra and some of its Consequences,' &c., which Dr. Maclean of Netley published in the 'Proceedings of the Hyderabad Medical and Physical Society,' the author stated that, with the exception perhaps of hydrocele, there is hardly any important surgical affection so common among the natives of that part of India in which he was then stationed (Hyderabad) as stricture of the urethra. This he attributed to the great prevalence of gonorrhœa amongst them and, above all, to the violence of the remedies employed for its cure. A common one, he was assured, is a strong solution of copper in some milk, which they inject, at all stages of the disease, into the urethra by means of a small bladder to which a quill is attached. Dr. Maclean had seen as many as six cases of stricture in one week.

In 1881, there were 5 admissions for stricture in the Hyderabad Contingent, and 11 in the native army of Madras.

I think that, for the last forty years, it has been generally admitted that nearly all the worst evils attendant upon urethral stricture are factitious—the handiwork, I will not say of surgeons (although in the early part of my career I saw much of urethral surgery which it pains me to remember), but of dressers and unqualified practitioners, to whom the boring of false passages appeared to come as instinctively as burrowing does to rats and foxes. I am not sure that it is at all to

the disadvantage of natives with stricture that theirs is a disease with which their hakeems, in many parts of the country, do not meddle instrumentally.

Of late, a good deal has been said about *Catheter Life*.

When a student, I watched a case which afforded a very remarkable type of that phase of existence. A fine muscular young man lived for many months with fracture of the lumbar spine. The catheter was regularly passed. When the urine began to be ammoniacal, Mr. Key told his dresser to inject half a pint of olive oil twice in the 24 hours, after emptying the bladder. He added, 'It will have a very soothing effect.' After some weeks of this 'soothing' treatment, we had an opportunity of examining the parts. The catheter, entering and penetrating the membranous portion of the urethra, passed along a sub-way *behind* the prostate, and entered the bladder through its trigone! This had evidently gone on for many days.

Urethral Fever probably depends upon a variety of causes—thus,

(1) Mr. Stafford long ago recorded an observation which, as coming from a London observer, affords a very important link connecting the surgical fevers of India and Europe. He said 'Ague very frequently occurs, from local injury, when the patient has, previously, had an attack of it. An injury of the urethra is a good example of this.' He adds, 'I have seen irritative fever, attendant on accidents, run on into intermittent fever.' Sir Joseph Fayrer has shown that urethral fever occurs in certain cases in India and other localities where there is a tendency to intermittent fever. Here anything which causes shivering—a chill or the passage of a catheter, excites a paroxysm.

(2) In confirmed Renal Disease,—*Morbus Brightii*, or Surgical Kidney, the introduction of an instrument acts as a stab would do.

(3) Septicæmia may be induced by the use of an unclean instrument. I long ago heard a conversation, in the shop of an eminent instrument-maker, to the effect that the mechanician would not take back certain catheters of his own making, which were declared to be as good as new. He said—'We used to take back and exchange such instruments; but, upon splitting them up, we generally discovered that they were full of blood.'

(4) In the late discussion upon Sir A. Clark's observations, it was repeatedly noticed that where, in cases of enlarged prostate, the urine is healthy, its condition speedily alters for the worse upon the commencement of 'catheter life.' I doubt much if this is primarily due to the use of the catheter. *It probably occurs because catheterism has been employed too late.*

DISEASES OF THE MALE ORGANS OF GENERATION

At page 493 of my 'Medical Jurisprudence for India,' I have given cases which show that, in that country, surgeons may expect to meet with cases in which the male genital organs are partially or entirely cut off for various reasons—viz. religious fanatics now and then inflict such mutilation upon themselves; clumsy circumcisors make dangerous wounds; the parts are removed by professional emasculators, or as a punishment for intrigue, and also for witchcraft. My notice of the latter fact, at p. 493, led, a few years ago, to the detection of the murderers of a wizard who, having been thus mutilated, was thrown over a precipice in the Kasia Hills.¹ The following notices do not appear in my book. In 1871, Dr. J. O'D. Partridge reported² the case of a man at Bustee who deliberately cut his penis off, leaving a stump about an inch and a half long, because a person refused to pay him a debt. He threw the amputated piece into this man's shop. A fakeer, of Nursingpore, was brought to Mr. Galt³ and said that, thinking that the lusts of the flesh prevented him from attaining a higher degree of sanctity, to which he aspired, he determined upon mortifying them; for which purpose, on the preceding morning, he laid what he regarded as 'the offending member' on a stone, and, with some sharp instrument, endeavoured to cut it off and cast it from him. His attempt was followed by such a hæmorrhage as caused syncope, in which state some of his neighbours found him. The wound was covered by a great clot of coagulated blood. The penis was greatly swelled, and no urine had been passed since the previous morning. Being in great pain, he requested Mr. Galt, for the sake of both his soul and body, to complete what he had begun, which from the probable injury of the great blood-vessels, &c., the surgeon feared he might be compelled to do, and had everything prepared for amputation if necessary. A catheter was, with difficulty, passed about half way down the urethra; its withdrawal was followed by some coagulated blood and urine. Not wishing to disturb the injured vessels by removing the clot, he bound up the penis with lint and plasters. After two days, it was found that urine escaped from a large wound in the dorsum penis and from another gash in the course of the urethra which had opened that canal freely. With some difficulty, a catheter was passed into the bladder, and was kept there for several days. The lower wound soon healed by the first intention, the upper one by suppuration and granulation. He left quite well. Returning some weeks afterwards to pay his respects, he said that the only inconvenience

¹ *I. M. G.* for October 1878.² *Ibid.*, vol. vi. p. 36.³ *I. M. J.*, vol. ii. p. 92.

he suffered was that of being cut by his reverend brethren until such time as, by giving them a *burra kana* (feast), he should atone for the offence by which, as they asserted, he had disgraced his cloth.

The following, from Ewart's Catalogue, describes the condition of parts in the case of a Mohammedan boy, *æt.* 11, of which a notice is given at p. 495 of my work. 'Prep. 884: an incised wound extending the whole way round the prepuce. On the upper surface, it is superficial, but it is deep on the under aspect, dipping down into the corpus spongiosum—not, however, penetrating the urethra. The boy is supposed to have died from the hæmorrhage which followed an unsuccessful attempt at circumcision. (For Tetanus, following circumcision, see p. 416.) One of my Mohammedan colleagues never heard of a fatal or dangerous case.

In 1784, Hyder Ali, having made prisoners of Colonel Baillie and a large body of his European force, had many of his prisoners circumcised under the influence of '*majum*,' a preparation of Indian hemp.

It appears, from the Report of the Administration of the Police of the North-Western Provinces for 1872, that Col. Tyrwhitt, Deputy Inspector-General of Police, considered that the offence of emasculation was then practised largely in those Provinces. 'There are,' it is added, 'many emasculators who are watched by the police.'

In my '*Medical Jurisprudence*,' there is a chapter on '*Epicenes*,' in which I have collected several observations on those spoken of by St. Paul, as being 'born eunuchs'—victims of congenital deficiency of the anterior wall of the bladder and symphysis pubis, or perhaps of extreme epispadias or hypospadias—who are in great request as the custodians of zenanas.

I give in that book facts which appear to throw some light upon the natural law under which the multiplication of these wretched beings, in India, may possibly come into effect. A few additional data are noteworthy. Sir James Simpson observed¹ that it is interesting to remark that, in some instances, malformations of the genital organs, giving rise to appearances of hermaphroditism, have been observed to be hereditary in families, both on the maternal and the paternal side; and, in some cases, to occur among several of the children of the same parents. Thus, Henremann mentions an example of a family, the females of which had, for several generations, given birth to males who were all affected with hypospadias; and Lecat alleges that a degree of hypospadias is not uncommon among families in Normandy. In '*Rust's Magazine*,' an instance is related of a degree of hypospadias existing in father and son. Baum, in his essay on congenital fissures of the urethra, has referred to two instances of the existence of hypospadias in brothers. Sir E. Home found two cases of hypospadias in first cousins. Kauw Boerhave mentions an example of four hypospadiac brothers,

¹ *Obstetric Memoirs and Contributions*, vol. ii. p. 347.

and Lepechin another instance in three. Naegele reported a case in which male twins were both hypospadiac; and Katsky and Saviard have mentioned similar instances. Mr. John H. Morgan cites¹ an article wherein Mr. Lingard gives an instance in which this deformity was traced through six generations, and relates a case in which it was transmitted by the widow of a hypospadian to four sons of a second husband who had no deformity. Mr. Morgan adds that he has, in several cases, been able to trace an hereditary transmission of this condition.

Preparation 889, in the Cal. Med. C. Museum, displays 'double penis, removed from a child which had also imperforate anus. The rectum terminated in the fundus of the bladder. There is a globular mass, underneath the scrotum, which may possibly be a superfluous scrotal bag.'

Dr. A. A. Mantell has described² the condition of a native, aged about 50, who appears to have been generally regarded as a female, but who seems to have been a male with an extreme degree of hypospadias. There were testicles and a penis-like organ, 1 inch 7 lines long, surmounted by a glans half an inch in length. At the top of the glans, was a sulcus or rudimentary meatus urinarius. An inch below this body, was what was regarded as a rudimentary vagina, half an inch broad; into it, a female catheter and a probe would pass for two inches, but to no greater depth; through this opening, the urine was voided in drops, and always with some little difficulty. Menstruation had not occurred, the penis-like organ was not capable of erection and the existence of sexual desire was denied.

A very closely similar case, in an individual who was also considered to be a female, is reported by Dr. J. Murray.³ A native prisoner, *æt. cir.* 30, was accused of taking liberties with another prisoner, a female. There was no hair on the face;—broad make, with strong muscular development, mammæ undeveloped. A very much enlarged 'clitoris,' more than an inch long, and exactly like a small penis, save that there was no orifice in the glans. At its root, there was a cutaneous pouch which contained one testicle about the size of an olive. On fully separating the labia, the meatus urinarius was found to occupy much its usual situation, but there was no other opening or canal of any kind, and not a trace of a vagina. 'She' positively asserted that she menstruated every two months by the urethra. This statement was, however, unsupported by evidence of any kind.

For *Epithelioma and other Malignant Diseases of the Male Genitals*, see *Epithelial Cancer*, p. 358.

¹ M. T., 'Clinical Lecture on Diseases of the Urinary Tract in the Young.' M. T., vol. ii. for 1885, p. 42—citing *Lancet*, April 19, 1884.

² I. M. G., vol. i. p. 3.

³ *Ibid.* for 1868, p. 207.

Preps. 256, 7, 8, and 9, 887 and 88 represent Epithelioma of the Penis and Prepuce; 260 Epithelioma of the Scrotum. Prep. 316 represents '*Fungus Hæmatodes* of the Prepuce and Glans Penis. The surface of the growth is lobulated and fissured. It is covered with many dark-coloured spots, indicating the points at which the extravasation of blood had occurred in the substance of the tumour. The surface is partially broken down by ulceration of the cancerous tissues.' A case of *Encephaloid Cancer of the Penis* occurred in the General Hospital, Madras, in 1882.

The late Dr. N. B. Baillie described¹ two cases of epithelial cancer of the penis in which, under the application of equal parts of acetic acid and water, as suggested by Dr. Wilks, the sores assumed a healing appearance and decreased in size. The final issue is not reported. We know that although, in these cases, the diseased surface may clean, the case still goes on to a fatal issue. I saw a case in which Sir J. Fayrer removed the diseased penis with the *écraseur*.² The specimen is numbered 256 in Ewart's Catalogue. 'An epithelial cancer from the penis of a Hindoo, aged 24, from the district of Burdwan. On microscopical examination, it was found to consist of many nucleated cells, free nuclei, fibres, and laminated cells' (Ewart). Not a drop of blood was lost in the operation. There was no marked cachexia, but the glands in the groin were slightly enlarged.

Malignant disease of the penis is of rather frequent occurrence in India, especially in Madras. Dr. Kenneth McLeod had ten cases of amputation, for epithelioma, in five years. There were eleven natives upon whom this operation was done in the General Hospital, Madras, in 1883. I do not find mention of such disease in the returns of the S. C. for the four years ending 1883.

I have spoken of *Idiopathic Sphacelus of the Scrotum* (death commencing at the scrotum), and *Elephantiasis of the Scrotum*, under the headings of Malarial Cachexia and Cholera, at pp. 256 and 296. I speak there also of *Elephantiasis* of the prepuce (*vide antea*, p. 263). Dr. Esdaile described a case in which the hypertrophied preputial integument was 'as large as an arm'; and Mr. Minas removed, at Mozuffernuggur,³ a preputial mass sixteen inches long which, when separated, weighed ten pounds. The scrotum remained unaffected.

Dr. Herbert Baillie has observed⁴ that, where hypertrophied skin has been removed from the penis, either alone or in conjunction with a scrotal tumour, the cicatrix which surrounds the root of the penis is liable to contract and so to constrict the organ as to render it incapable of being distended, and, in course of time, to cause it, from want of proper nutrition, to shrivel into a button-like body. This

¹ *I. M. G.*, vol. ii. p. 44.

² *I. A.*, No. 16, p. 298.

³ *I. M. G.*, vol. viii. p. 214.

⁴ *Ibid.*, vol. iv. p. 259.

inconvenience may be obviated by making, at the time of the operation, one or two straight cuts, at least two inches long, upward, radiating from the circular incision, and of the same depth, through the healthy integument of the pubic region. By this contrivance, much the same result is gained as the tailor obtains in letting in a gusset; for the incision gapes and its edges eventually adhere at a little distance from the original stellar line of incision; and thus the constriction of a circular cicatrix is as it were eased off.

Piliferous Cyst of Scrotum.—Sub-Assistant-Surgeon Cowasji Pestonji describes the case of a Hindoo broker (age not given), the subject of congenital scrotal hernia, who came with abscess of the scrotum. ‘A well-curved tuft of hair, about the size of a hen’s egg, escaped, leaving a deep cavity.’ The cavity filled up, but a fistulous opening remained.

Hydrocele of the Cord.—Preparation 28, Series 12, Madras Collection. A native was admitted, by Dr. J. Liston Paul, with a tumour [in the inguinal canal] which, for the last five years, had constantly descended into the scrotum. About twelve days previously, the tumour increased in size and became painful. There was a hot and painful tumour in the groin; percussion elicited a tympanitic sound, with a feeling as if of contained bowel. Great abdominal pain, with tenderness on pressure. Bowels had not acted for five days. Purgative enemata brought away a quantity of faecal matter. Under chloroform, the tumour could not be passed into the abdomen, although it was considerably diminished in size by taxis. Although the symptoms of strangulation were not urgent, it was decided, in consultation, to cut down on the neck of the tumour. It was found to envelope the cord, which could not be felt, and to descend through the external ring. The sac was opened and the tumour appeared to be composed of a series of small cysts. It lay upon the epididymis and testicle, from which, however, it was readily separated, and then drawn up to the neck, where it was found to have two attachments, one running along the cord as far as the internal ring, the other taking an upward and forward course, towards the iliac fossa. On traction being made, Poupart’s ligament was moved. A ligature was applied to the neck of the tumour, which was cut off below the internal ring. It consisted of a fleshy mass holding numerous small cysts. The patient died of acute peritonitis on the following day.

The late Mr. Cutcliffe¹ treated a healthy-looking Hindoo, *æt.* 30, who had a *Hydrocele of the Tunica Vaginalis connected through the inguinal canal with a large cyst situated within the abdomen.* This condition was treated effectually by repeated iodine injections.

I had a native patient in whom there was obstinate constipation with symptoms of constitutional distress. A hernia-like swelling, of

¹ *I. M. G.*, vol. viii. p. 124.

the size and form of a hen's egg, occupied the inguinal canal on one side. I consulted Sir Joseph Fayrer, and we decided to cut down upon it. It proved to be a hydrocele of the cord.

Dr. Maitland has also described a case of hydrocele of the spermatic cord complicated with symptoms of strangulated hernia.¹ Preparation 96, p. 147, in the Madras Museum, apparently represents this case. A fibro-cystic tumour removed from the left groin. The patient was admitted with symptoms of strangulated hernia. The history indicated hernia. The tumour was, however, quite immovable, dense and resistant, and no impulse was communicated to it on coughing. The part was very tender. The symptoms being very urgent, the parts were cut down upon, but no hernia could be found. A small cyst protruded through the ring and communicated with the tumour. The latter was removed, the cyst was ligatured and pushed back. Recovery without a bad symptom. The walls of the cyst were intimately blended with the cremasteric fascia, and its dissection was difficult. Its contents, besides serum, were a dark-coloured gelatinous material and bands of recent lymph.

To this remarkable class of diseases evidently belongs *Orchitis of Malarial Origin*. A native practitioner of Berhampore, a malarious place, wrote² a suggestive but rather loosely-reasoned paper on *Periodical Orchitis*, known in Bengal as *akshera*, as it usually attacks only one testis. He says that, in some localities, the majority of the native inhabitants are affected, and that the inflammation 'recurs about every full and new moon.' His description evidently includes cases of incipient scrotal elephantiasis. He attributes the affection to gonorrhœa. Most persons will doubtless think, with the editor of the 'Indian Medical Gazette,' that it is an expression of paludal cachexia. All who have served in Lower Bengal have doubtless seen cases of this kind; but, as far as I am aware, no other observer has noticed the great prevalence of the disease in certain districts.

See the following notes on *Hydrocele*.

Hydrocele.—I had a native patient whose death was caused by acute inflammation of the tunica vaginalis. This was probably a phase of elephantoid fever. It is here noteworthy that '*Neuralgia of the Testis*' is not a frequent entry in the returns of the S. C. In the four years ending 1883, two European and eight native soldiers were treated. This organ is doubtless liable to simple neuralgia; but I think that, where Indian cases of idiopathic orchitis and neuralgic testis are sifted, it will generally be found that, especially in Lower Bengal, when the suffering is great and chronic, there is more or less inflammatory action present, tending to the development of either hydrocele or elephantiasis scroti, or of both.

¹ *I. M. G.* for 1882, p. 210.

² *I. M. G.*, vol. i. p. 213.

I have already spoken of hydrocele under the heading of 'Malarial Cachexia,' page 254.

Fayrer's 'Clinical and Pathological Observations,' p. 513, contain a valuable chapter on the 'Malarious Origin of Hydrocele.' The disease is very common in Lower Bengal, principally among the very poor, much more so than would be expected, judging from the statistics of the sepoy and jail hospitals of India in the three years ending 1882, which give: sepoys, 72, 54, and 34 admissions; prisoners, 112, 115, 47; the average strength of the former class being 118,630, and of the latter, 100,556. It is clear that, with higher constitutional power, the sepoy is less liable than the prisoner, and I think that a considerably larger proportion of cases would be found among the poorest Bengalis. The disease is frequent in rich natives and in Europeans who have long done heavy work in India. Had this disease been of less prevalence in that country, it is probable that we should not now have occupied India. In 1715, the E. I. Company began evidently to give way under the exactions and oppressions of Moorshed Kuli Khan, the Nawab of Bengal. It was therefore determined to send an embassy to the Emperor Furruckshere, at Delhi, with a view to obtaining recognition of their old firmans and immunities. Against the fulfilment of this project, all the craft and energy of Moorshed were directed, with every prospect of success, when, happily, the intervention of Mr. William Hamilton, who had accompanied the embassy as their surgeon, rescued the Company from their difficulties. The marriage of the Emperor with the daughter of Raja Ajeet Sing, one of the Rajput princes, had been for some months delayed in consequence of the fact that he suffered from a hydrocele which his own physicians were unable to cure. The princess had arrived at Delhi, and matters were assuming a very serious aspect, when, by the advice of the Khan Dowrah, who had become the patron of the embassy, Mr. Hamilton was consulted, operated, and was so fortunate as to cure the Emperor. Delighted at his recovery, the Emperor heaped gifts and promises upon the surgeon, who is known to have accepted, at a durbar, among other presents, a robe of honour, a culgi set with precious stones, two diamond rings, an elephant, a horse, gold buttons for coat, waistcoat, and breeches, set with jewels, 500 rupees, *and models of all his surgical instruments in pure gold*. When desired to name his reward, his sole entreaty was that the Emperor should grant the requests of the ambassadors. Consent was freely and immediately conceded to the objects of this generous, self-denying mediation; and,—although after considerable delay attendant upon the marriage festivities, and prolonged by the active machinations of the Nawab's agents,—a firman was granted, confirming all the original privileges of the Company, permitting their president to grant passports exempting goods from search throughout

Bengal, allowing them the use of the mint at Murshedabad, and permitting them to purchase thirty-eight additional towns (villages) near Calcutta, at a rent of 8,121½ rupees (812*l.*). The Court of Directors, doubtless not being fully informed with regard to Mr. Hamilton's action, desired, in a despatch to Calcutta of February 1715, that he should be dismissed on his return. A Court order of January 1716 revoked Dr. Hamilton's dismissal, because he had cured the Great Mogul. The Company did not receive their firman until 1717—in the unhealthy autumn of which year good William Hamilton was carried to his grave, in the old cemetery by the Fort Green. His beautifully carved tombstone is still preserved. I published an exact copy of it.¹ When Warren Hastings opened St. John's Cathedral, he walked out and visited Hamilton's tomb, and desired that its finely cut raised inscription and ornaments should be gilt, and the slab be placed within the sacred building. I made a private attempt to have this order carried into effect thirty years ago, but the tomb is still outside, in Charnock's monument. If Walpole judged rightly that 'every man has his price,' it is to be wondered at what rate this poor surgeon would have forfeited his integrity!

In old times, hydrocele, as well as many other tumours, was often allowed to attain an enormous size before surgical aid was sought. This doubtless occurred in the Emperor Furruckshere's case. One of Ranald Martin's early patients, who was brought to the Calcutta Medical and Physical Society, was relieved of nearly a gallon of fluid, after which the scrotum 'hung between his knees like a bheestie's bag, —the tanned sheepskin used by the water-carriers.'²

Prep. 873 in the Calcutta Medical C. Museum is a tunica vaginalis, presented by Mr. S. B. Partridge, from which 182 ounces of fluid were removed. The patient, an old man, died from diarrhœa contracted in hospital.

In his valuable 'Miscellanea Medico-Chirurgica,' Mr. Hussey describes a case of hydrocele, in which the scrotum was as large as the patient's head, as by far the largest hydrocele which he has seen.

Fayrer has remarked that the hypertrophy of the fibrous tunics of the testicle is sometimes very remarkable. He has seen the tunica vaginalis thickened to the extent of half or three-quarters of an inch, and the dense fibrous tissue is sometimes the seat of cartilaginous or even ossific deposits. Preps. 138 to 141, 870, 872, 877, and 878 represent this change in progress. I saw a remarkable example of this rare condition in one of Aston Key's patients in Guy's Hospital. The surgeon, who justly prided himself upon the possession of a *tactus*

¹ In an article on 'Surgeons in India—Past and Present,' in No. 44 of the *Calcutta Review* for September 1854, and again in the *I. A.*, No. 4, p. 716.

² *I. M. J.*, N. S., vol. i. p. 235.

cruditus, said, 'Weigh and press this testicle.' I did so; it was as hard and heavy as a very small cocoanut, and of about the same size. He added, 'This is a specimen of that very rare disease, scirrhus of the testicle; but, in such cases, we always take the precaution of exploring. Give me a sharp bistoury.' The knife appeared to grate, as if passing through cartilage, and then a spirt of limpid fluid sprinkled the operator.

In my student days, Mr. Earle's very dangerous plan of injecting port wine was generally practised in England; but, as early as April 5, 1834, the late Sir Ranald Martin presented to the Medical and Physical Society of Calcutta his 'Notes on a new Injection for the Cure of Hydrocele,'¹ in which he reported that he had injected two urethra-syringes-full of a solution of tincture of iodine, in the proportion of 2 drachms to 6 of water of the ordinary temperature, in upwards of 90 cases, without any danger or (surgical) inconvenience whatever. It was shown, in a subsequent report, that, up to the middle of 1838, Martin had operated, at the Calcutta Native Hospital, on 1,381 cases, with only 6 failures. Attempts by other surgeons to cure by a weaker solution had not been successful.²

When I left India, the strong tincture was usually employed. I saw cases in which tapping sufficed, especially that of an aged medical officer who used to tap his own hydrocele. He perfectly recovered, moderate inflammation having supervened upon the second or third puncture.

The late Mr. Cutcliffe described³ the case of a Baboo, who, having had the contents of a hydrocele evacuated by a Kobiraj,—who scratched the skin and then applied a plug of wood, coated with some escharotic, which ulcerated its way into the sac and allowed its contents to escape,—suffered from a purulent discharge for five months and then sought aid with a scrotum rather larger than a man's head. In the centre of the front (right) of the scrotum were two ulcers, through which a probe of double length went straight to the back of the scrotum. Here was a honeycombed and irregular cavity pouring out a horribly stinking fluid, mixed serum and pus. Daily injection of iodine which was, at last, twice as strong as the tincture of the British Pharmacopœia, produced steady contraction of the mass and healing of the ulceration in three months.

Preparation 31, Series 12, in the Madras collection, is a tunica vaginalis from a European who died from acute dysentery in a week after having a hydrocele injected with tincture of iodine. 'The recent specimen,' says Mr. Sibthorpe, 'was most interesting. The lymph appeared like a mass of clear jelly and the walls of the sac were injected.'

¹ *Transactions*, vol. vii. part 1, p. 204.

² *I. M. J. N. S.* vol. iii. p. 421.

³ *I. M. G.*, vol. iv. p. 238.

A case of Hæmatocele of the Cord has been reported by Dr. Hodder.¹

Hæmatocele.—The first surgical case which I had to treat in India was that of a native prisoner with a very large hæmatocele; but this condition is not common in India. In the three years ending 1882, only 8 sepoys and 5 prisoners were treated. Many years ago, a most worthily distinguished administrative medical officer took the very unusual course of reporting that a surgeon had erred in not being able to distinguish a case of — from one of hæmatocele. Ultimately, the senior frankly declared himself to be in error.

A case is recorded by the late Mr. Cutcliffe² in which an aged native with hydrocele received a blow on the tumour, which was followed by increasing acute pain about the external ring and great swelling. There being distinct fluctuation at the lower part of the tumour, about a pint of clear serous fluid, quite transparent and free from all traces of blood, was withdrawn. A circumscribed hardness remained at the upper part of the scrotum. The pain continuing to be very severe for some days, the upper tumour was cut down upon and proved to be a hæmatocele containing about six ounces of dark clotted blood. The cyst of this was situated over the anterior surface of the epididymis (globus major) of the testicle, and did not extend to its lateral and under surfaces. It appeared that a septum had formed and divided the tunica vaginalis into two parts.

Hæmatocele is illustrated in Preps. 874–76 in Ewart's Catalogue.

Dr. Kenneth McLeod has reported³ an interesting case of what appears to have been *Sympathetic Orchitis*. A Mohammedan, aged 30, suffered from abscess of the left mammilla, which was opened but did not discharge freely. In six days, he became feverish, and his left testicle inflamed and became about as large as a fist. As the abscess healed under treatment, the testicle gradually regained its normal dimensions. Having carefully eliminated every other possible cause of the orchitis, the reporter was compelled to the conclusion that it was caused or determined by the irritation of the mamma on the opposite side.

Preparation 18 in Ewart's Catalogue displays 'extreme *Atrophy of the Testes* in a Hindoo boy, æt. 16.' They seem to be mere flattened expansions of the cords, being about three-quarters of an inch long and a quarter of an inch thick.

In a considerable proportion of cases, sexual power is restored upon recovery from ablation of elephantoid scrotum; but, in some instances, especially when deep sloughing has been caused by injudicious native practice, the structure of the glands suffers. Prep. 880 in the C. M. C. Museum represents *Atrophy of the Testicles*. One

¹ *I. M. G.*, January 1875.

² *Ibid.* vol. iv. p. 262.

³ *Ibid.* vol. iv. p. 237.

is extremely attenuated; the other evinces considerable hypertrophy of the tunica albuginea. From a case of elephantiasis scroti.

In Prep. 17, there is 'atrophy of the testes from the pressure of two adventitious growths in a case of elephantiasis scroti.'

In 1880 a sepoy was admitted with atrophy of a testicle. There was another case in 1882. A European soldier was treated in 1883.

Prep. 883 displays *Tubercular disease of the Testes*. Unfortunately the race has not been specified in this and in the cases of scirrhus, (710). 'Circumscribed deposits of tubercular matter are seen. These are of a brown yellow colour, whilst those of the stroma of the organ are of a dull opaque white.

A chapter of Sir Joseph Fayrer's 'Surgery in India' is devoted to *Fungus of the Testicle*. The author employed, with great success in many cases, Syme's operation of releasing the gland from constriction, and freeing the integument sufficiently to allow it to be brought over the growth, and then of uniting the edges of the scrotum over the tumour, retaining them in apposition by suture.

Dr. Herbert Baillie remarked¹ that instances not unfrequently occur in which either the patient objects to submit to Syme's operation, or where it and pressure have failed. Here, if the testicle be much constricted by the surrounding tissue, this should be first divided by a few stellar incisions; then the testicle and parts divided are to be thickly covered with burnt or desiccated alum, retained by strips of soap plaster; and, over this, a carefully applied bandage. The loose alum should be removed daily, and fresh applied, and well pressed down, that which adheres and cakes being allowed to remain till it falls off, when it may be renewed. In addition to this, it is well now and then to pour tincture of iodine over the salt. Generous diet and preparations of iodine with iron are needful. The cure is generally complete in three weeks or a month; but, where the protrusion is very great, double that period may be required.

I had great faith in the efficacy of Bransby Cooper's plan of daily inserting red precipitate all round the neck of the fungus where it and the scrotal integument meet.

Preparation 162 displays a multilocular *Seminal Cyst* situated on the external aspect of the investing membrane of the testis. Its fibro-cellular wall is not more than half a line in thickness. The agglutination of its posterior wall to the outer surface of the tunica albuginea is clearly demonstrated. Its interstitial surface is smooth and velvety.

Varicocele occurs in India, but not so frequently as might be expected in so hot a country. In the four years ending 1882, the annual admissions of men of the native army of India were only

¹ *I. M. G.*, vol. iv. p. 183.

5, 10, 4, and 8. The admissions of European soldiers were 18, 16, 28, and 30.

Preps. 881 and 882, C. Museum, are specimens of *Scirrhus of the Testis*. In the first, the structures of the cord and tunica albuginea are involved. The cancerous nodules springing from the testicle are well illustrated. In the second, scirrhus is not quite so advanced. It can be seen, however, to include the soft parts of the cord and the tunica vaginalis reflexa.

In a report on the 1st Troop Horse Brigade,¹ Mr. A. H. Leith gave the case of a man whose right testicle was forced into the inguinal canal when he was at sword-exercise. Afterwards, during severe straining, it passed into the abdominal cavity and remained there. The man denied ever having had hernia, and, a short time before the accident, Mr. Leith had strictly examined him previous to his being admitted to the Horse Artillery.

Sir Joseph Fayrer treated a case of *Medullary Disease of an Undescended Testicle*² in a Hindoo *æt.* 45. The testicle appears to have been arrested in its descent and to have remained quiescent in the inguinal canal, between the rings, until the middle period of life, when it began to increase in size and to be painful. It was hard, nodulated, and about as large as an orange. He had a somewhat thickened scrotum and also hydrocele. There was no testicle in the scrotum on the right side. The pain and swelling had commenced four months previously. The tumour was cut down upon and removed; the cord appeared quite healthy. The diseased testicle was an example of the firm kind of medullary cancer. The surrounding tissues were not involved, and, when he was discharged cured, there was no appearance of disease elsewhere.

In 1881 a sepoy was admitted with 'malposition' of a testicle.

Dr. Dantra has reported³ the case of a native writer, *æt.* 26, who had no outward appearance of testes, although he had suffered from three attacks of gonorrhœa.

DISEASES OF WOMEN.

In India, soldiers' wives, although subject to a very high rate of mortality, are mostly, on landing—for their class—strong and healthy women. The wives of officers and other gentlemen in that country are, almost invariably, before the climate tells upon their health, types of female strength and form. But a small proportion of sickly,

¹ *I. M. J.*, vol. ix. p. 659.

² *I. A.*, No. 16, p. 323, and *Clinical Surgery in India*.

³ *I. M. G.* for 1877, p. 299.

strumous, crooked, rickety, stunted, chlorotic, amenorrhœal women go to India, where aged Englishwomen are scarcely to be found. I believe, speaking from experience, although I have no statistical evidence to go upon, that, among European ladies in India, the diseases peculiar to women are, in the present day, less varied and less frequent than they are at home; but, in this and in every other respect, the barrack-women of India are very far less healthy than the soldiers' wives in the United Kingdom.

The only tables showing the annual death-rates of officers' wives and widows, are Davies's (for wives), and Neison's (for widows), both between the ages of 16 and 50. Wives, 15·85; widows, 12·28; women in England and Wales, 10·86 per 1,000 annually.

In the ten years ending 1838, the death-rate among soldiers' wives, in the exceptionally unhealthy station of Secunderabad, was 63·59 per 1,000. Mr. Hugh Macpherson found that, in the five years ending 1854-55, the death-rate among this class, in Bengal, averaged 44·5.

Ewart shows¹ that, from 1871 to 1875, the death-rate of the wives of European soldiers in India was 25·19 per 1,000; and that, from 1876 to 1880, it was 23·67. The Sanitary Commissioner's report for 1880 gives a mortality, for all India, of 21·05 *per mille*. In Bengal, it was 22·64, in Madras, 17·31, and in the Bombay Presidency, 20·75. The men's death-rate in all India was 28·32. In 1881, the death-rate of soldiers' wives, in all India, was 25·93 *per mille*. In the Bengal Presidency, it was 29·30, in that of Madras, 18·89, and in that of Bombay, 24·60. In that year, the death-rate of European soldiers throughout India was 16·86. In 1882 the death-rates of soldiers' wives were, in all India, 19·78; in the Presidency of Bengal, 21·57, Madras, 13·81, in that of Bombay, 22·19. In all India, the European soldiers' death-rate was 12·07.

In 1883, when the soldiers' death-rate, in all India, had fallen so low as 10·88, that of the wives was 21·28; Bengal, 21·44, Madras, 16·71, Bombay, 27·30.

In the United Kingdom, between 1877 and 1881, the death-rate among soldiers' wives ranged from 7·75 to 11·06 per 1,000 annually (Marston and Ewart). Ewart further shows that more than half of the mortality among this class in India, or about 12·667 per thousand, is caused by a few diseases of tropical climates. He gives the ratio of deaths in India from diseases peculiar to women as 3·39 *per mille*.

I have long thought, and have urged in public, that, in every Indian station, a lady,—not a mere nurse,—of tact, resolution, and judgment, should be officially authorised to look after these women, who are generally young, inexperienced, and absolutely imprudent in all matters

¹ 'Causes of Excessive Mortality among Women and Children of the European Soldiers serving in India,' *Transactions of the Epidemiological Society of London*, 1882-83.

of hygiene. It once came within my official experience that a hideous mortality occurred among soldiers' children, mainly in consequence of the mothers' folly and ignorance.

At a meeting of the Calcutta Medical and Physical Society, in 1835, Dr. Duncan Stewart observed that most of the young females who came out to India had menstruated before embarkation, but it very frequently happened that they did not menstruate above once during the voyage; and that, before the healthy re-establishment of this function, a painful series of nervous disorders has to be undergone. This was in the days of the long sea-voyage. I believe that those who go to India by the overland route do not incur these evils.

There is a great deal of sexual disease among native women. Child-marriage and premature maternity, the varied atrocities committed by native midwives, hard work, poor food, the frequent practice of criminal abortion, often work together in producing disease for the relief of which skilled aid is too frequently rejected. It is shown in my 'Medical Jurisprudence for India,' p. 667, that suicide, attributed to various causes, among which disease stands prominently, is very frequent among native women. Dr. Shortt, of Madras, a physician well acquainted with native customs, states¹ that he has reason to believe that the great majority of suicides among native women is brought about by uterine diseases; for perhaps after years of silent suffering, and unable any longer to endure the agonising pain, aggravated by the deterioration of the general health, the unfortunate female takes relief in suicide. He adds: 'The accounts we frequently read after coroners' inquests in which the suicide is frequently attributed to *colic*, is, I think, a misnomer on the part of the friends of the deceased, who, in giving evidence, withhold the true cause that led to self-destruction, or the word *uddy viarathu novoo*, pain in the lower belly, is mistranslated into *colic*.'

Writing of Marwar in February 1873, Mr. P. H. Hindley said:² 'The amount of suffering in zenanas is hardly conceivable. Many women are broken down at a very early age by uterine disease, dyspepsia, and affections of the eye—the latter nearly all the effects of neglected conjunctivitis. It would be a boon if female medical missionaries could be found ready to devote themselves to alleviating this lamentable suffering.'

In an interesting pamphlet entitled 'Medical Women for India,' Dr. Frances Elizabeth Hoggan gives the following particulars. The Maharajah of Punna, Central India, applied to Miss Beilby, a lady medical missionary at Lucknow, for medical aid to his wife, the Maharanee, who had long been suffering from a painful internal disease, for which native custom precluded her from seeking relief at the hands of

¹ *Madras Journal of Medical Science* for January 1871.

² *I. M. G.*, vol. viii. p. 40.

a male doctor. On recovery under Miss Beilby's care, the Maharanee entreated that lady to convey a message to Queen Victoria representing the sufferings which the women in the zenanas in India endure when they are sick. She explained that it was no social change in their condition she sought, but relief in their cruel sufferings.

In Lower Bengal at least, there is probably scarcely one poor native woman in fifty who is free from intestinal worms—in itself a grave cause of suffering and ill-health, and an aggravating complication of other disease. I have already mentioned that, in my female ward, every woman and child had infusion of quassia as a vehicle for all fluid medicine. This very cheap and excellent vermifuge and tonic ought to be taken daily in every native household. Dr. Waring has observed¹ that he does not think the statement exaggerated that eight-tenths of the women of Mergui are labouring under either *Amenorrhœa* or *Dysmenorrhœa*. He considers that, without doubt, this is to be attributed, in a great measure, to their indiscriminate use of the cold bath twice a day, under almost all circumstances, excepting during the presence of intermittent fevers. The presence of the catamenia forms no bar to the daily use of the cold bath; injunctions and arguments are of no avail in preventing this practice. In this respect, they differ from the Hindoo women, who abstain from bathing for three days at every menstrual period. Still, the ordinary practice of native women in Lower Bengal of bathing, defecating, and washing their thin cotton garments without taking them off, in tank or river, and of then walking home laden with a full water-jar, their wet drapery clinging to them in the chilly morning air, cannot but be productive of disease.

The painful details given in my 'Medical Jurisprudence,' pp. 688–700, afford strong evidence to show that the native child-wife enters upon her matrimonial existence under physical disadvantages scarcely to be imagined by the European mind.² The frequent practice of criminal abortion, which I have also very fully illustrated (p. 712), in all its various forms, each exceeding the other in atrocity, must, when it does not kill, lay the foundation of much disease and suffering.

None of those modern gynæcologists to certain of whose proceedings Dr. Matthews Duncan objects so courteously and yet so validly, are more addicted to the local use of instruments and medicaments than the native *daees*—midwives—are. Sub-Asst. Surgeon Syud

¹ 'Medical Notes on the Burmese,' *I. A.*, No. 1, p. 98.

² It is quite needless to repeat this painful detail here; but Preparation 954 in the College Museum is sufficiently illustrative. It displays 'laceration of the perineum and a considerable portion of the vaginal sheath, the effect of violence, on a Mohammedan child of 12, in the first act of copulation. She appears to have been found dead from hæmorrhage.

Mahomed Jann placed on record¹ a case which affords us a very slight insight into native practice. A native woman of Jhundy, *æt.* 24, having aborted six or seven years previously, having subsequently remained barren, and wishing to become a mother, took various kinds of native medicines with that view. She sent for a native midwife, *machin*, who advised her 'to introduce a bit of *katore fruit*, cut in a certain shape and of a certain length, into the canal of the cervix during menstruation, and to have intercourse after a certain length of time, having previously removed it. Such a connection, she thought, in ninety-nine cases out of a hundred, would be fruitful.' The effect, of course, was local inflammation and fever. When removed, the foreign body was more than three-quarters of an inch in length, and about the thickness of a goose-quill.

So Ambrose Paré has it, *Caput IV., de Hominis Generatione*: '*Juvabit ante . . . indidisse in uteri cervicem et osculum moschi aut zibethæ tantillum.*'

There is a great deal of *Debility* among European women, rich and poor, in India. Among the wives of European soldiers, averaging 3,804, throughout India, there were, in 1880, no less than 1,036 admissions and 6 deaths under this heading. In the following year, there were 1,100 admissions, with 3 deaths. In 1882, the admissions were 991 and the deaths 3. In 1883, the admissions for debility were 2553, and the deaths 98 *per mille* of strength. In 1880, the admissions of soldiers' wives for *Anæmia* numbered 17. In 1881 they were 29; and in the following year 48. In this year, 2 cases of *anæmia* proved fatal. In these three years, only one case of *Chlorosis* was admitted—in 1881. No case appears in the return for 1883. It would hardly be too much to say that nearly every native woman in India is more or less *anæmic*.

To this low state of health must be attributed the premature sterility of natives and the necessity which compels nearly every English lady to seek strength at home at least once in every decennium. These influences often lay the foundation of those disorders of function and organic lesions which are attended with *Amenorrhæa*, *Leucorrhæa*, and *Menorrhagia*. The returns give, in the four years ending 1883, the following admissions of soldiers' wives, for *Leucorrhæa*,—23; 21; 34; and 44. For *Menorrhagia* there were 46; 39; 59; and 35. The admissions for *Amenorrhæa*, during this period, were 7; 8; 17; 17. In all probability, the returns do not represent the full amount of suffering from these morbid conditions.

I saw much of *Menorrhagia* both in Europeans and natives. In the very severe case of a young married East Indian woman, a native *dacc* had made matters far worse by inserting a plug into the cervix. In consulting with a very efficient practitioner who had been well

¹ *I. A.*, No. 6, p. 702.

taught, but had not 'qualified,' I said, 'If you do not succeed in checking the menorrhagia your patient will die.' 'Yes,' was the reply, 'I said to her, "Mrs. — your amen-norr-hœa is so dreadfully profuse that, unless you take my advice, you will probably bleed to death!"' For a note on *Intermittent Menorrhagia*, see Malarial Cachexia, page 250.

Mr. E. C. Bensley, the editor of the 'Indian Medical Gazette,' and Baboo Ram Lall Dey have written upon the use of Indian hemp in menorrhagia, and of uterine hæmorrhage generally, but they agreed in thinking that, here, the drug is apt to cause attacks of syncope. Dr. Churchill, quoted by the Baboo, speaking of the curative effects of Cannabis in menorrhagia, said, 'I have once or twice seen unpleasant nervous symptoms follow its use, but they were not serious, and were dissipated in a few hours by ammonia, rest, &c.' The editor remarks 'There can be no doubt that such effects as are described by Mr. Bensley not unfrequently follow the administration of Cannabis. We should say that a patient taking this remedy ought carefully to be kept recumbent.'¹ I have not used Cannabis in menorrhagia, but I have never considered that it had a depressant action in the diseases for which I employed it. On the other hand, fainting occurs, from loss of blood, in menorrhagia where Indian hemp is not employed.

Dr. Harvey has reported² the case of a native, æt. 25, suffering from a growing fibromyoma of the uterus. There was menorrhagia causing anæmia, weakness, and loss of flesh. Oöphorectomy was performed and the patient was reported to be convalescent.

Leucorrhœa.—Many years ago, I treated several cases of *Idiopathic Vaginitis*, in children. Subsequently, I did not happen to see anything of this condition. The following note occurs in the old Madras Medico-Topographical reports:³—'European children are subject to a discharge from the vagina on attaining their seventh or eighth year. The fluid is commonly of a muco-purulent appearance; and at times is in such quantity as to affect the general health, inducing weakness, wasting, and loss of appetite; sometimes pain is felt about the uterine region, with uneasiness and scalding in voiding the urine.' I looked upon the 'weakness, wasting, and loss of appetite' rather as the concomitants than as the consequences of the local affection, which generally yielded without much difficulty to cleanliness, good food, and quinine and iron. It does not appear that this disorder is constantly prevalent among the soldiers' children. The returns show that, in 1880, there was one case of leucorrhœa treated in all India. In 1881, there were three admissions under this heading; in the following year, one child was treated for vaginitis; and, in 1883, there was only one admission.

¹ *I. M. G.*, vol. i. pp. 175 and 256.

² *Ibid.* for January 1884.

³ For Madras, p. 90.

Dr. Joseph Ewart has reported cases of *Uterine Catarrh* of long standing which he treated successfully in Calcutta upon Dr. Playfair's system of applying the remedial agents in a fluid form to the interior of the cervical and uterine cavities by means of swabs.¹ Both authorities place especial reliance upon carbolic acid; thus, acid carbolic 4 parts, glycerine 1, water 5.

Dr. Ewart had considerable experience of the vast utility of carbolic acid, either in its pure liquid form or somewhat diluted with glycerine and water, in cases of *Excoriation*, *Ulceration*, or *Granular Inflammation of the margins of the Os Uteri*, so common in Calcutta.

The late Dr. H. H. Goodeve obtained the following mode of treating leucorrhœa from a native midwife. A muslin bag, like the finger of a glove, filled with a mixture of strong astringents, such as equal parts of powdered alum, cinchona bark, and catechu, is temporarily introduced.

I consider that I met with a very few cases of what is termed *Vicarious Menstruation*—i.e. discharge, from sources other than the uterus, of blood, or hæmoglobin associated with deranged menstruation. Dr. Duncan Stewart² gives the case of a Frenchwoman who had alarming hæmoptysis. This 'was found to be connected with menstrual obstruction and enlarged spleen. The discharge continued three days. During this time, she brought up two pounds of blood. Two relapses had occurred. For a day or two before the bleeding began, the spleen became enlarged and felt heavy and painful, causing nausea and faintness when the patient walked.' It must here be recollected that, in India, women who have great splenic hypertrophy or confirmed malarial anæmia are generally *amenorrhœal*.

I have more than once seen plum-juice expectoration in women who had no organic pulmonary or cardiac disease. One of these patients, an East Indian lady, whom I attended more than thirty years ago in such an attack, and in whom there was no sign of heart obstruction, and no concurrent evidence of tubercular or other lung mischief, came to England a few years ago, and is, I believe, still residing in my neighbourhood.

Dr. W. J. Elmslie has recorded³ the case of a plump healthy-looking Kashmiri girl, *æt.* 13, who first menstruated scantily at the age of 10. At the third period, the natural discharge was very slight, but blood in small quantities flowed from the bowels, ears, nose, and mouth. Neither fright, chill, nor any other cause could be assigned for this irregularity. When seen, she said that she felt well, although menstruating. There then flowed from her ears and nostrils a thinnish dark-coloured fluid, which was in small quantity but was slightly coagulable. Every now and then, she spat saliva tinged with blood.

¹ *I. A.* No. 21, p. 149.

² *I. M. J.*, vol. iii. p. 47.

³ *Ibid.* vol. ii. p. 103.

Her mother stated that then, as formerly, a small quantity of the discharge came by the vagina, and a little also by the rectum.

In No. 8, N. S., of the 'Bombay Medical and Physical Transactions,' Mr. D'Andrade published a case 'in which the menses were apparently substituted by hæmorrhage from the skin.'

While I am quite in accord with Dr. Wilks in considering that the full function of menstruation cannot be performed vicariously, it is certain that there is a tendency to oozings of hæmoglobin or blood, from ulcerated and other surfaces in women in whom menstruation is not normally performed.

Among the women of European regiments in India, averaging 3,741 in number, there was registered, in each of the three years ending 1882, only one case of *Cancer*. Two are noted as fatal. There were three cases, one fatal, in 1883. The larger proportion of these women, however, have not reached the cancer age.

Considerable *Congenital Malformations of the Female Organs of Generation*, have been rather frequently observed in India. Prep. 940 in Ewart's Catalogue is chiefly remarkable for the imperforate penis-like clitoris.

In the report for 1883, a labour at the Madras Lying-in Hospital was rendered complex by the existence of a *Bi-partite condition of the Uterus*.

Dr. J. Ferguson observed¹ the following condition in a native woman, *æt.* 22, who, although married seven years, was childless, who drowned herself. 'There were two distinct orifices and two distinct and separate uterine cavities, each communicating with its corresponding Fallopian tube. The vagina was normal.'

Imperforate Hymen.—In 1882, Dr. Wallace brought to the notice of the Medical Society of Calcutta,² the case of a child, nine months old, in which there was a sulcus but no vaginal orifice.

A native girl, *æt.* 16, was brought to the Cawnpore dispensary greatly debilitated and emaciated, with a sallow complexion, fever, and an anxious expression of countenance. Evacuation of the bladder and bowels was attended with pain. The abdomen was swollen to the size of the gravid uterus in the fifth month, vagina closed by a smooth, red membrane (the hymen) distended and pushed outwards, as if by the descent of a child's head during parturition. An opening being made, exit was given to three quarts of a chocolate-coloured fluid. The abdominal tumour subsided and the girl felt instant and complete relief to all her sufferings. She appears to have done well. The first symptoms of the accumulation seem to have been noticed four months previously.³

Dr. Charles had a similar case;⁴ but the race of the girl is not

¹ *I. M. G.* for October 1874.

² *Ibid.* for that year, p. 137.

³ *I. M. J.*, N. S., vol. v. p. 487.

⁴ *I. M. G.* for 1867, p. 229.

mentioned. There was a large abdominal tumour which was considerably lessened but not wholly removed by drawing off forty ounces of urine. On dividing the hymen, which was imperforate, and a quarter of an inch thick, eighteen ounces of a treacle-like fluid were evacuated. The uterus was found to be very slightly enlarged, but the vagina was greatly hypertrophied.

A tall, thin, native girl, *æt.* about 18, was treated by Sub-Assistant-Surgeon Ashootosh Goopto at Delhi.¹ She had never menstruated, but, from the age of sixteen, had been liable to temporary attacks of pain in the loins and uterine region. For the last four months, there had been a swelling above the pubes, which was found to be about as large as a foetal head. It was painful and tender, and there was an expression of great suffering when the thighs were flexed over the abdomen. The vagina was occluded, at the depth of about an inch, by 'hard tissues of a whitish colour.' After exploration, a large trocar was passed through the partition, which was three-fourths of an inch thick. Two or three pints of tar-like, bloody fluid escaped. On the following morning, Dr. Adam Taylor cut through the obstructing tissue, which was hard and almost cartilaginous, and introduced a thick pledget of lint. Moderate abdominal pain and tenderness, with feverishness, followed. The vagina admitted three fingers. She was discharged well nine days later.

Dr. W. D. Stewart found *Occlusion of the Os Uteri* in a Hindoo woman of the Nutt or gipsy caste, *æt.* 23, who had never menstruated, but had been married three years. A dimple indicated the position of the os. On puncture, a large quantity of dark thick fluid flowed away. No bad symptom followed.²

In a report on Kashmir,³ Surgeon-Major G. C. Ross gives the case of a young married native woman treated for occlusion of the os uteri. An actual cautery was forced in where the os should have been, and half a gallon of dark menstrual fluid escaped. Copulation had been effected by the urethra; consequently, there was incontinence of urine which, when she left the dispensary, had become relieved by contraction of that canal.

In 1883, Dr. Branfoot employed the thermo-cautery knife in a case of retained menses which proved fatal.

Prep. 52, Series 13, in the Madras Collection, is from a girl *æt.* 15. Uterus with vagina and portion of the rectum, showing atresia which was congenital and caused retention of the menses. A small exploring needle was passed by the rectum, when some dark menstrual fluid escaped. Death from peritonitis occurred four months later.

My friend, Dr. S. C. Sarbhadicary,⁴ attended a [native] lady who thought that she was about to miscarry in the eighth month of

¹ *I. M. G.*, vol. ii. p. 246.

² *Ibid.* January 1874, p. 13.

³ *Ibid.* for October 1878.

⁴ *Ibid.* for 1867, p. 229.

pregnancy. He discovered no foetal heart sounds or other signs of pregnancy. The catheter could not be passed. After waiting a fortnight, he introduced an exploring needle and then a trocar into the tumour, and drew off twenty pints of treacle-like fluid. The tumour gradually decreased in size, but the patient died a fortnight after the operation. No *p. m.* was obtained. She had menstruated regularly till within eight months of the operation. It is not improbable that this obscure case is explained by that which follows.

A healthy, well-formed girl, about 19 years of age, came to Mr. A. Morgan¹ at the Bancoorah Charitable Dispensary, stating that, about two years previously, she was delivered of her first child, which was born without any difficulty and lived a few days; but, the placenta being retained, the *daee* who assisted, in order to cause its expulsion, introduced a quantity of the juice of the 'Lalkhosidia,' or shoe-plant, into the vagina, which caused very severe inflammation, on the subsidence of which she found that the orifice had become closed. On separating the external labia, no vaginal orifice could be seen, the labia minora being joined together by, and themselves absorbed in the substance of a fleshy cicatrix, in the centre of which was a small opening capable of admitting a probe, and which led nearly in a straight line to the os uteri, the canal of the vagina being apparently obliterated with the exception of this passage, through which the catamenia flowed. The cicatrix was carefully and gradually divided, partly with the knife but chiefly with the fingers, until the os uteri was reached; by this means, a spacious vagina was formed and was then filled with oiled lint. No bad symptoms followed, but the canal was disposed to contract. The narrowing which had taken place, about an inch from the orifice, was incised, a bougie was used and she was discharged well towards the end of the second month.

In the Hospital and Dispensary under the control of Dr. Branfoot at Madras, three cases of atresia vaginæ were admitted in 1882. Two of these were operated upon, both successfully. In 1883 Dr. Branfoot reported two more cases of the same condition.

In the four years ending 1883, the numbers of European soldiers' wives treated for *Dysmenorrhœa* were 11, 28, 17, and 30.

Dr. Soorjee Coomar Sarbhadicary, of Calcutta,² believes that 50 per cent. of Bengali women suffer from dysmenorrhœa. He considers that it is dependent in most cases on mechanical obstruction of the os uteri. The bark of the root of the oolut kambul (*Abroma angusta*, N.O. Sterculiaceæ) is often successful in these cases when they have resisted all other treatment. It is given in scruple doses with the same quantity of pepper (*gol mirch*), every day for two months. Baboo Bhoobun Mohun Sircar has since very strongly recommended

¹ *I. A.*, No. 13, p. 329.

² *I. M. G.*, vol. ii, p. 229.

the use of oolut kambul in dysmenorrhœa.¹ Dr. Chuckerbutty thought it likely that this drug owed its reputation for efficacy rather to the stimulant action of the pepper mixed with it than to any properties of its own. He attributed the prevalence of dysmenorrhœa in India to the frequent exposure of women to cold and wet, when bathing or washing their feet, as they always do after evacuating the bowels.

At a meeting of the Calcutta Medical Society held in February 1881, Dr. David B. Smith related the case of a young woman, suffering from this disorder, who had exhausted the air out of a brass *lotah* (this is a very strongly-made water vessel having a wide mouth, a narrower neck, and a large fundus—hence the difficulty) by burning a small oil lamp in it, and placed the vessel on the lower part of the abdomen for the purpose of dry-cupping the part. The abdominal walls entered the cavity of the *lotah* and could not be withdrawn until the bottom of it was removed. There were not any unpleasant results. Dr. Bowser said that this mode of dry-cupping is common. It is called *guti boshun* and *gar boshun*. Tumeez Khan Bahadur and Kanny Lall Dey Bahadur had seen similar cases in which holes had to be drilled in the bottom of the *lotah* before it could be removed. Dr. Naylor related a case in which, though the top had been removed, the pot could not be taken off. It had to be sawn through. Baboo B. B. Chakravati said that dislocation of the navel—‘*Nar Khola*’—is assigned as a cause of abdominal trouble of various kinds and is treated in this fashion. Baboo B. M. Sirkar remarked that earthen vessels are now used for this purpose.

Dr. Joseph Ewart has reported² two cases of *Conical Cervix Uteri* in young European women. Dr. Branfoot treated seven of these cases, in natives, in 1883.

I have discussed the cases of *Prolapse of the Uterus* which are so common in India (generally the result, in women of all races and classes, of imprudence in beginning to go about too soon after delivery) in speaking of the *Diseases of Pregnancy*.

In the four years ending 1883, the admissions of soldiers' wives for prolapsus uteri were 23, 21, 15, and 12.

Uterine Flexions.—The views of the only two Indian gynæcologists of the present day, with whose opinions I am at all acquainted, are, I believe, more or less strongly in favour of the conclusion that flexions of the uterus are states of disease and frequent causes of suffering in European, East Indian, and native women. Still, in the quadrennial period so often cited, the numbers of European soldiers' wives, admitted for *Displacement of the Uterus*, were only 1, 6, 3, and 2—cases of prolapsus being separately returned. Dr. Charles remarked, in a discussion on dysmenorrhœa which took place at a meeting of

¹ *I. M. G.*, vol. vii. p. 153, and vol. viii. p. 98.

² *Ibid.* vol. viii. p. 208.

the Bengal Branch of the British Medical Association,¹ that membranous dysmenorrhœa depended very frequently on follicular ovaritis; he had often been able to tell patients, in whom he found tenderness over the ovaries, that they suffered from painful menstruation with discharge of membrane, and their answers had confirmed his assertion. He added, 'Anteflexion or retroflexion of the uterus often caused dysmenorrhœa, which ceased when the womb was replaced, or the inflammatory symptoms were combated.'

In his Report for 1882, Dr. Branfoot gives the following data. Out of 109 Europeans and East Indians, treated as in-patients, there was one case of anteversion; of anteflexion, 9, of retroversion 10; and retroflexion 14. In 167 natives, there were 7 cases of anteflexion, 9 of retroversion, and 12 of retroflexion.

In 1883, among 115 Europeans and East Indians treated, there were 12 anteflexions, 16 retroversions, and 7 retroflexions. In a total of 188 natives there were 1 case of anteversion, 7 of anteflexion, and 8 of retroflexion.

In Bengal, within the influence of the sea breeze, native women are liable to huge *Elephantoid Enlargements of the External Organs of Generation*. As I have already mentioned (p. 268), Allan Webb considered that, when such hypertrophy attacks the nymphæ, there has been syphilis. Sonatun Bysack removed² a tumour, involving the mons and right labium, which weighed $8\frac{1}{2}$ pounds. The history points to the probability of syphilis.

In the case of a poor unhealthy native married female, about 25 years old, who was an outcast from her home, Dr. W. A. Green³ removed a tumour as large as a child's head, which depended as far as the knee from the mons and right labium. Its lower part was ulcerated. She died, in a month after the removal of the mass, from phthisis with bowel complication.

Sub-Assistant-Surgeon Ram Eshuar Awasthee⁴ operated upon a native woman, æt. 25, in whom both labia were affected. When removed, they weighed 12 pounds. She was discharged well in less than a month.

I have a note of a case in which a tumour of the same kind, $11\frac{1}{2}$ inches long and 25 inches in circumference, was successfully removed.

In the case of a Mussulmaunee, æt. 30, Dr. J. E. T'uson⁵ removed, quite successfully, an elephantoid tumour from each labium. The two together were four feet in circumference.

Ram Eshuar Awasthee⁶ described and figured the case of a Mohammedan woman, aged about 22 years, in whom a rounded tumour which reached as far as the middle of the thigh, arose from the glans

¹ *I. M. G.*, vol. ii. p. 229.

² *Ibid.* vol. viii. p. 67.

³ *I. M. J.*, vol. ii. p. 309.

⁴ *I. A.*, No. 2, p. 585.

⁵ *I. M. G.*, January 1874, p. 14.

⁶ *I. M. J.*, N. S. vol. iv. p. 534.

of the clitoris. It had been growing for three years. It so pressed upon the orifice of the urethra that she was obliged to lift it up with her hands when making water. It was removed, and the wound healed by the first intention.

Several specimens from cases of this kind are preserved in the Madras M. C. Museum. A syphilitic origin is repeatedly traced.

Dr. Branfoot's return of cases treated in the gynæcological wards of his hospital at Madras in 1882, gives five cases of elephantiasis of the labia and clitoris. There were five cases also in 1883.

The eight preparations, numbered 196 to 199, and from 946 to 949, in Ewart's Catalogue, represent enormous elephantoid growths from (1) the *labia majora*, and (2) the *labia, nymphæ*, and *clitoris*. Chinese women are liable to enormous growths of this description.

A writer from Poonah in the 'Indian Journal of Medical and Physical Science'¹ said that a healthy European female came under his care for a large *Abscess in the Vagina*. It had a tedious suppuration and was several weeks in cicatrising. He learned that, at the same time, about ten women, living in the same locality, had been similarly affected. They attributed the disease to the bad water of the place.

Preparations 941-42 in Ewart's Catalogue represent *Ulcers of the Vagina*.

The following histories deserve attention in relation to Dr. Max Graefe's recent paper on *Vaginal Cysts*.²

Mr. Scales³ narrates the case of a European woman, *æt.* 45, who came to India in her tenth year. When seen she had been married twenty-one years; a tumour had appeared about a month afterwards. It was of the size and shape of the top of a small mushroom, and, at first, caused no pain or inconvenience. When she had her first child, it increased a little, but went on very slowly for years. At the birth of her last child, about eleven years afterwards, it first protruded externally and became of a dark colour. After that, it grew much faster and caused great uneasiness. The tumour was about as large as a turkey's egg, pear-shaped, and protruding from an inch to two inches from the vulva. It was quite insensible to the touch. It was attached, beginning close to the orifice of the urethra, to the whole anterior part of the vagina and to the cervix, but not to the os uteri. Another smaller growth, about the size of an olive, was attached high up, both to the larger tumour and to the vagina. There was, at intervals, intense pain in the region of the uterus, with the sensation of great bearing-down and a constant discharge of blood-like matter. She was very weak and anæmic. On account of its large attachments, the

¹ N. S. vol. iii. p. 409.

² *Zeitschrift für Geburtshilfe und Gynäkologie*, and *M. T.*, p. 31, vol. i. for 1884.

³ *Madras Quarterly Medical Journal*, vol. iv. p. 177.

tumour was first punctured. About an ounce of clear gelatinous fluid was let out. The tumour decreased to the size of a large marble. She continued to improve in health for ten days, when, having taken a great deal of exercise, she suffered severely from pain and bearing down, and the tumour was found to have increased to about half its former size. The tumours were then removed; they contained a gelatinous fluid. The reporter says 'the walls of the tumour were very firm and of a fibro-cartilaginous structure'—the growths were evidently cysts. There was, for about ten days, 'a discharge of gelatinous matter without any pain.' She made a good recovery, and was quite well in a month.

Mr. Branfoot removed vaginal cysts successfully in two cases in 1883.

A native prostitute applied at Dr. Hutchinson's dispensary with a cystic swelling of the left labium, about the size of a hen's egg, of three months' standing. The passage of an exploring needle gave vent to two ounces of an extraordinary fluid of a rich brown colour, and irritant withal, as the hand sensibly smarted under its influence. The microscope revealed nothing but pigment-granules with a few epithelial scales here and there.¹

Prep. 38, Series 16, in the Madras Collection, is a fibro-cystic tumour removed from the left labium of a married woman. 'The interior simulates the cauliflower excrescence.'

In India, *Pruritus Vulvæ* occurs as in temperate climates. I repeatedly met with cases in middle-aged women.

In 1873, my friend Dr. Joseph Ewart published² a valuable practical commentary on the treatment of *Vaginismus*. It is founded on the cases of four married women. Two of these were perfectly unaware of any disability prior to marriage. In both, the painful condition made its appearance immediately afterwards. In one, sexual congress was quite impracticable on account of the spasmodic pain produced by the extraordinary contractions of the sphincter vaginæ and the hyperæsthetic state of the outer surface of the remains of the hymen and of the mucous surface extending from this to the fourchette. In the other, it was tolerated, but was always accompanied by much pain and hysterical agitation of the nervous system. The painful part included only a small portion of the mucous membrane on each side of the caruncula myrtiformis and covering the fourchette. It was well-defined, congested, red and shiny, and excessively sensitive. The remainder of the mucous membrane at the outlet was healthy in colour and in no way participated in the hyperæsthesia. One of the women had been married five years, the other two years. Both were childless. A third patient had been married

¹ *I. M. G.*, vol. iv. p. 117.

² *I. A.*, No. 31, p. 141

about five years, and, during the whole of that period, excepting the first few days after marriage, she had been unable to bear sexual intercourse on account of the agonising pain with which it was accompanied. The vagina could not bear the passage of the finger. The mere touching of the outlet threw the patient into a state of general spasm, every muscle in her body appearing to be all-attention and in a state of extreme tension, simulating the convulsion of tetanus. The caruncula myrtiformis presented the usual appearance throughout three-fourths of its circumference, extending on each side backwards from the orifice of the urethra. The remaining fourth, on the posterior aspect, was intensely vascular, shiny and smooth, as was also the mucous membrane of the fourchette and fossa navicularis. The hyper-sensitive condition was absolutely confined to the vascular spot, and was localised to a surface about the size of a sixpence on each side of the median line, and including a corresponding breadth of the external surface of the caruncula myrtiformis. The condition of the fourth patient was in all respects identical with that of the third.

The principle of Dr. Ewart's operation is that the diseased mucous surfaces alone were excised, and the wound, being brought together by deep and superficial sutures, was placed under the most favourable conditions to secure union by the first intention. After this, dilatation was had recourse to for a limited period. He deprecates Sims' deep Y-shaped secondary incisions as tending to impair the retentive muscular power of the vagina. Dr. P. Lewis examined the excised structure. He found it '*extremely* vascular, the nerve-filaments quite superficial, and the subcutaneous papillæ bare.'

I have spoken of *Noma Pudendi*—*Malarial Phagedæna*, as it occurs in splenic cachexia, at p. 258. It is displayed in Prep. 953 of Ewart's Catalogue but, in that subject, syphilis and mercury were also at work.

Mr. McGann relates ¹ two cases of *Eversion of the Urinary Bladder* in women of middle age. See Diseases of Pregnancy.

A case of this accident, in a soldier's child in the Bengal Presidency, appears in the return for 1883.

Epithelioma of the Os came several times within my observation in Bengal. In Dr. Branfoot's wards, Madras, in 1882, there were five cases of cancer of the uterus in 109 European and East Indian patients, and the same number in 167 native patients. The cervix uteri was amputated in two of these cases, and scraped and cauterised in another. In one case, the uterus was removed for carcinoma. In 1883, this gentleman had two cases in Europeans and eleven in natives, in one of whom the uterus was removed *per vaginam*. She died on the second day.

¹ *I. M. G.* for February 1, 1879, p. 46.

Among Dr. Branfoot's patients, there were, in two years, two cases of *Polypus* in natives and one in a European. A few cases of uterine polypus in natives came within my observation in Bengal. Preps. 927 to 931 represent polypi growing from (1) the os and (2) the uterine cavity. Preparation 931 exhibits a small almond-shaped polypus springing by a narrow pedicle from the immediate neighbourhood of the opening of one of the Fallopian tubes. It was not attended with any symptoms during life. Unfortunately, the races of the women are not specified.

Specimen 106, S. 13, in the Madras Collection, is a mucous polypus growing by a thin pedicle from the mucous membrane of the cervix uteri of a young native woman who had been amenorrhœal for months. It was easily removed by avulsion. Its surface is covered with a layer of inflammatory lymph, and sections display a large number of mucous crypts in a fibrous stroma, hæmorrhage having occurred into some of those near the surface. It occasionally came outside the vulva.

Ram Eshuar Awasthee¹ removed by ligature from a Mohammedan woman, *æt.* 40, a huge polypus attached to the os uteri by a pedicle thicker than a thumb, and forming a tumour protruding from the vagina.

Atrophy of the Uterus is represented in 'C.M.C.' Preps. 909–10.

Fibroid Tumours are of rather common occurrence. Preps. 932–36 illustrate this form of non-malignant growth. Prep. 937 is 'A fibrous tumour of the uterus, globular in shape, and as large as a man's head.'

In 1882, three Europeans and East Indians and five natives were treated for fibroid in Dr. Branfoot's Madras institutions. In the following year, he treated seven cases in Europeans and four in natives—one fatal.

Ovarian, Dermoid, Piliiferous, and Dentigerous Cysts.

Prep. 165 in Ewart's Catalogue is an ovarian cyst with several tumours springing from its internal surface. It contains a quantity of *hair*, which is lying in detached balls, and agglutinated to the parietes. Prep. 168 presents two cysts, as large as hens' eggs, springing from the right ovary. They contain *hair and a soft cheesy substance*, consisting of oil-globules and epithelial scales. The dense fibrous character of the parietes of the growth, now distended with the material, is well demonstrated; the floor of that which has been almost emptied of its contents is formed by this hairy and fatty matter. The section shows that the remainder of the ovary is honeycombed by primitive cysts varying from the size of a pin's head to that of a bean. From the upper surface of the left ovary, several small cysts spring, resem-

¹ *I. A.*, No. 2, p. 586.

bling nothing so much as a cluster of Cabool grapes. The subject was probably a native. Prep. 169 is from a woman, *æt.* 27. Before the operation, the ovarian tumour measured nineteen inches vertically and twenty inches transversely. The abdomen measured three feet seven inches in circumference. During the operation, the upper and back part of the tumour was found to have undergone malignant degeneration, which involved some of the adjacent viscera, from which a mass of encephaloid, as large as a child's head, was removed. The patient sank sixteen hours after the operation. The growth was first noticed three years previously, but it had remained stationary till within the last ten months, during which it attained its large size. Ewart observes that the specimen is of peculiar interest, owing to its large size and its consisting of several loculi, characters seldom presented by this variety of ovarian tumour. When emptied of most of its contents, except the almost solid *fat* of one of its compartments, which was about the size of a child's head, it weighed 19 lbs. 4 oz. Some of the cysts are barren, others proliferous; *hair and teeth are embedded in the fatty substance.*

Dr. Cayley¹ treated an ovarian piliferous cyst, as large as a man's head, containing fifty-eight ounces of semi-fluid matter, in a Hindoo woman of 40, by injecting three drachms of a mixture of one part of tincture of iodine to four parts of water. She had no unfavourable symptoms, was able to go home in nineteen days, and had no signs of return of the disease in nine weeks.

I have known *Prolapse of the Ovary* to occur in Bengal in the European and in the Eurasian.

I treated a case of *Acute Oöphoritis* in a European. In the returns for the four years ending 1883, the numbers of European soldiers' wives admitted for Ovaritis were 6; 7; 4; and 9, one fatal.

Webb's Prep. 597 displayed *Atrophy of the Ovaria.*

Prep. 12 in Ewart's Catalogue is '*Atrophied Ovaries.*' They are flattened and attenuated. Neither exceeds the twenty-fourth of an inch in thickness.

Prep. 145 is '*Section of an ovary showing Calcareous Degeneration of its stroma.*'

Prep. 890 represents *Abscess of the right Ovarium*, which opened into the fundus of the bladder. It is probably from a native patient.

Ovarian Tumours occur in women of all races in India—the available statistics do not show how frequently. In the four years ending 1883, there were, among the soldiers' wives, only two cases.

Preps. 899 and 900 '*C.M.C.M.*' illustrate *Malignant Disease of the Ovary.*

The eight Preps., 891–98, illustrate *Ovarian Cysts*, simple and multilocular.

¹ *I. M. G.*, vol. i. p. 218.

Prep. 166 is an ovarian tumour consisting of one large cyst, from the interior of which several pedunculated tumours are suspended. The right ovary is considerably enlarged, owing to a large proportion of nuclei and granules in its stroma.

Prep. 167 is a large multilocular ovarian tumour, consisting of several small cysts, varying from the size of a hazel-nut to that of an orange. One of the parent cysts is as large as a man's head. The walls are very thick, dense, and strong.

Prep. 1,113 is a multilocular ovarian cyst from a woman, *æt.* 30. The abdomen measured forty-five inches in circumference. The tumour measured forty-five by twenty-five inches. From the time of its being first noticed, it took about four years to reach this size. The cysts were tapped through one puncture. Decomposition of the remaining portion of the contents of one of the cysts took place, apparently hastening death, which took place in two days.

Dr. J. M. Joseph¹ removed large ovarian tumours successfully in two native patients at Combacum. Dr. W. P. Dickson performed the operation in a young Hindoo woman, who made a favourable recovery.² Dr. R. C. Sanders also had a successful case of ovariectomy in a native woman of 20.³ Dr. Charles performed several of these operations, at some of which I was present, at the Calcutta Medical College Hospital. In 1882, Dr. Branfoot, of Madras, treated four Europeans and East Indians, and 6 natives, at his hospital for ovarian cystic disease. Of the former patients, one was operated upon and recovered; the tumour was removed in five natives, one of whom died. The cases are fully detailed in the superintendent's report. In 1883, Dr. Branfoot treated one case in a European and five in native patients. In six cases the diseased growth was removed. There were four deaths. The cases are reported at large.

Dr. J. O'Brien successfully removed, in the case of a Khasia woman, aged 25, an enormous ovarian tumour—a very large central cyst with numerous smaller ones. The mass, with its fluid contents, weighed 42 pounds.⁴

Dr. Willcocks, Civil Surgeon of Benares, has lately reported⁵ nine cases of ovariectomy in native women. Seven were successful. At the end of that year,⁶ a surgeon of Amritsur published the case of a Hindoo, *æt.* 20, who was married at 11, and had conceived twice. The abdomen was very tense and its circumference was five feet four inches. The tumour adhered on all sides. It was made up of three large loculi filled with gelatinous purulent fluid, and many smaller cysts. It weighed six pounds and its fluid measured several pints.

¹ *I. A.*, No. 9, p. 274.

³ *I. M. G.* for November 1877, p. 292.

⁵ *I. M. G.* for 1885.

² *I. M. G.*, vol. ii. p. 123.

⁴ *Ibid.* for November 1880.

⁶ *Ibid.* for December.

Webb's Prep. 206 represents *Occlusion of both Fallopian Tubes*, cystic degeneration of the ovaries, and atrophy of the uterus in a native prostitute.

Prep. 901 in the C. Museum Catalogue displays great *Dilatation of the right Fallopian Tube*. Its internal surface is roughened and irregular. The left tube is also dilated and internally much roughened by inflammatory deposit. Its fimbriated extremity is adherent to the ovary. The cavities of two recently enlarged and ruptured Graafian vesicles open directly into the attached fimbriated channel of the tube. From a patient who died from excessive hæmorrhage from the uterus. This appears to have been a case of hæmo-salpinx.

Prep. F 32 in the Grant M. C. M., Bombay, displays 'expansion of the Fallopian tube into a cyst about the size of an egg.'

Prep. 47, Series 12, in the Madras M. C. Collection, shows the Fallopian tubes closed and dilated at the outer extremities. Prep. 48 'shows two cysts, one within the other, and both attached to the extremity of the right Fallopian tube. There is no communication between the cavities of the cysts and the canal of the tube.'

In the body of an East Indian girl of eleven, I found a white unilocular *Cyst*, as large as a cricket-ball, attached to the *Broad Ligament*.

Prep. 63, Series 12, in the Madras Collection, is a simple cyst of the right broad ligament, removed by gastrotomy from an unmarried woman, *æt.* 17 years. It had commenced two years previously, and was attended by considerable pain on the right side. It was extensively adherent to the omentum and intestine, and was separated with difficulty.

I have twice seen enormous *Distension of the Urinary Bladder* in native women. In the first case, a patient in my own ward, I at once perceived the nature of the swelling and passed the catheter. In the second, which I saw in consultation, I was absolutely wrong. Dr. Charles was not to be deceived by one or two (probably erroneously) alleged circumstances in the case which misled me.

Pelvic Hæmatoma, Abscess, and Peritonitis, unconnected with Pregnancy.

The following case was recorded ¹ by the late Dr. Simpson, Civil Surgeon of Dacca. 'A Mohammedan female, *æt.* 25, menstruated at 12, became pregnant at 13 years, and was delivered of a male child. Since that period, has not been pregnant, but has menstruated regularly, and without pain or disturbance, except a tendency to menorrhagia. Has always been rather thin and delicate. States that, about twenty days ago, she menstruated as usual; and, on the fourth day, was suddenly seized with sharp pain in the hypogastrium, and became feverish.

¹ *I. A.*, No. 13, p. 330.

The pain and fever increased daily, and she soon observed a swelling in the hypogastrium, painful on pressure, and increasing, progressively with the fever, up to the present stage. She is greatly emaciated, countenance indicative of intense suffering, lying on her back with knees drawn up, strong fever, quick small pulse, dry furred tongue, great thirst, retention of urine, constipation; general tumefaction of the hypogastric and iliac regions, with extreme tenderness on pressure, the slightest manipulation causing intense suffering; describes the pain as constant and throbbing.' A distinct, globular, smooth tumour, with a defined boundary, as large as the uterus of the fourth month of pregnancy, occupied the hypogastric region and pelvis. A considerable quantity of urine was drawn off. On vaginal examination, a pelvic tumour was felt behind the uterus. Os uteri high up, tilted forwards. A female catheter readily passed into the uterus; the dimensions of its cavity were normal. Passing the forefinger of the left hand into the rectum, and that of the right into the vagina, a large tumour was grasped, occupying the recto-vaginal space, hot and exquisitely tender, and giving a soft quasi-fluctuating feel. Pelvic cellulitis, advanced to suppuration, was diagnosed. At the most dependent part of the tumour, behind and below the os-uteri, an opening through the roof of the vagina into the tumour was made with a straight narrow bistoury, guided on the finger. Instead of the expected pus, blood, partly fluid and partly in clots, flowed out in considerable quantity. A soft coagulum of blood was found as far as the finger could reach. The wound was now enlarged by the finger and the bistoury, and, by gentle pressure, a portion of the contents was evacuated. The patient being much exhausted, further evacuation of the contents was not deemed prudent. A piece of lint was introduced into the wound, and folded cloth wet with cold water placed over the hypogastrium and vulva, and a sedative saline draught was ordered every four hours. On the following morning, the constitutional disturbance and local inflammation were aggravated. On removing the lint, there was free discharge of bloody fluid mixed with clots. Fomentations were ordered and the draughts continued, with full doses of morphia at night. On the third day, constitutional disturbance was undiminished; symptoms more 'typhoid' (typhus-like). Putrid sanious discharge, mixed with clots, escaped from the vagina. On the fourth day, the constitutional distress and local pain and tenderness began to be less, and the discharge of decomposed blood was abundant. On the three following days, the constitutional and local symptoms moderated; discharge free but less foetid. From the seventh to the fifteenth day, there was slow improvement. Fever, irritability of stomach, pain, tenderness, and hypogastric tumefaction diminished, and the discharge was more purulent and less foetid. On the fifteenth, there was a very copious discharge of pus mixed with clots. Convalescence was fairly established

and, under nutritious diet and wine, she rapidly improved. The wound was kept open by occasional dilatation with the finger. On the twenty-eighth day, the tumour had disappeared, the wound was healed, and the patient was daily regaining flesh and strength.

Dr. David B. Smith has narrated¹ a very interesting case of 'combined pelvic hæmatoma and pelvic abscess.' A Bengali woman, *æt.* 26, had had one child and had become a widow seven years before her illness. She had generally menstruated regularly. Nine days previous to her admission, the catamenia had come on with great pain in the uterine region. Before the discharge ceased, she noticed a swelling forming in the left iliac region. Intermittent fever, preceded by rigor, had been present for a week. When Dr. Smith first saw her, she was in great distress, with hot skin and rapid pulse. A large tumour was found externally, extending as high as the gravid uterus at the end of the sixth month. It was painful on pressure, firm and dull on percussion; no actual fluctuation could be made out anteriorly. On vaginal examination, a swelling was detected posteriorly. The uterus was pushed up quite beyond the reach of the finger. A uterine sound was introduced with considerable difficulty. The uterus was unimpregnated and of natural size. A considerable swelling, between the vagina and rectum, was detected. On the following day, fluctuation existing between the vagina and rectum, an incision was made through the posterior vaginal wall. About six ounces of a pinkish fluid, limpid and clear, evidently serum and blood, without coagula, escaped. There was no pus. The great bulk of the tumour remained as before, except that it was slightly collapsed over the *left* iliac fossa. The finger entered a chamber thoroughly emptied of its contents. On the following day, she was no better, and, in consequence of the urgency of the constitutional symptoms, an incision was made higher up through the wall of the chamber. About eight ounces of blood and serum escaped, still no pus. The tumour now somewhat collapsed over the *right* iliac fossa, but still it was nearly as large as ever, extending from pubes to umbilicus. The finger now passed into a second chamber which had also been quite emptied of its contents, but the roof of which could not be reached. After this, she suffered much pain in the right iliac region. The application of a blister greatly relieved this. The wound was kept open, and the tumour was constantly poulticed; she was well supported and took quinine. In four or five days, the fever began to return. Seven days after the last opening was made, she, whilst at stool, passed a large clot of blood and about an ounce of pus from the vagina. She was then in an alarming state, with great heat of skin, and rigors, pulse 120. With a good deal of difficulty, the finger was passed through the two incised openings, and a further pro-

¹ *I. M. G.*, vol. i. p. 150.

jection of the tumour downwards was reached. Dr. Smith proposed to puncture this, but the thin wall in contact with the tip of his finger gave way spontaneously, and more than a pint of very foetid pus escaped. The tumour now almost completely subsided. She became very weak, but was kept up by brandy and nourishment. It could not be ascertained where the opening into the rectum was situated. From this time, she continued to improve. The size of the tumour became gradually reduced until it altogether disappeared. The discharge diminished in quantity. There were two or three attacks of fever and a threatening of dysentery which yielded to treatment. The discharge had all but ceased when she was discharged, a month and fifteen days from the date of her admission.

Dr. Smith held that the cause of pelvic hæmatocele is very obscure, and that it still involves a debated point. The French school, for the most part, believe it to be always intra-peritoneal, and the result of the rupture of ovarian vessels, whilst the possibility of the existence of the extra-peritoneal variety is more commonly allowed by English writers, and this either from hæmorrhage at the menstrual period, from violent efforts shortly after menstruation or delivery, or from a varicose condition of the sub-peritoneal veins. Bernutz went the length of saying that an extra-peritoneal hæmorrhage was not a genuine hæmatocele, but simply a thrombus. In Dr. Smith's opinion, it is to be regretted that he endeavoured to institute any such distinction, and the voice of subsequent observers, particularly in England, is decidedly against such a nosology. He believed the present case to have been one of extra-peritoneal hæmatocele; this partly because the swelling extended between the rectum and the vagina, almost to the outlet of the latter, and again because he felt tolerably confident that he did not, in making his first incision, divide the peritoneum. He remarked that the fluid which escaped was almost exactly like that described by Dr. Matthews Duncan in certain cases recorded in the 'Edinburgh Medical Journal' for November 1862. It was a syrup-like blood. Whether, in this case, the pelvic abscess was intra- or extra-peritoneal, it was much more difficult to say. He was inclined to think it was the latter, although the existence of a large quantity of fluid in the posterior peritoneal *cul-de-sac*, commonly called Douglas's fossa, would help to simplify the explanation of the extremely retroverted condition of the uterus which existed in this case. Whether this abscess was the result of secondary inflammation, lighted up by the presence of the blood effusion, or whether the hæmatocele itself suppurated, as it is sometimes known to do, it was extremely hard to determine; indeed, these two points appeared to him the most difficult in connection with this case, viz., 1st, whether it was possible that the hæmatocele was extra-peritoneal, and the abscess intra-peritoneal; and, 2nd, what was the exact relation of the one lesion to the other in its nature and order of production. He inclined to the idea

that both were extra-peritoneal, and that the abscess was encysted in false membranes, whilst the hæmatoma was diffused in the cellular texture, behind and on both sides of the womb. To such tumours as surround the uterus, Bernutz has applied the term 'peri-uterine hæmatocele.' With regard to the diagnosis in this case, Dr. Smith holds that he was correct, inasmuch as there was pelvic abscess, the presence of which he suspected throughout. He adds that he failed to diagnose hæmatoma. In favour of its existence was the suddenness of its occurrence, the co-existence of menstruation, &c.; against it was the presence of fever, the painfulness of the tumour, and the hectic condition of the patient. Dr. Smith observes that his case (the same remark applies to Dr. Simpson's case) is interesting as disproving the opinion of Bernutz that hæmatocele is met with only in connection with pregnancy or the puerperal state. Dr. Smith's judicious remarks upon the puerperal and non-puerperal forms of pelvic cellulitis, especially as they occur in India, and upon their treatment, will be found under the heading '*Pelvic Cellulitis*' in the section on 'Diseases connected with Pregnancy,' p. 777.

The following case, by Dr. Branfoot (xlv. for 1882), appears to throw considerable light upon *pelvic hæmatoma, peritonitis, and cellulitis, as they occur in the non-pregnant state*. Rago, æt. 20, native Christian, no children, admitted in a low state, and died in six hours. Stated that she had not menstruated for three months and supposed herself pregnant. Ten days before admission, she had been beaten, and, three days after this, got severe pain in the abdomen with profuse bleeding from the vagina. Her periods were, as a rule, scanty, and attended with severe pain. She had a blanched appearance and an anxious expression of countenance. Pulse hardly felt at the wrist, heart's action very rapid, respiration short and hurried, temperature 98·4 F., lower abdomen distended and very tender. Passes frequent dysenteric stools. Vagina hot; cervix small, high up, jammed behind symphysis pubis by a fluctuating tumour bulging forward from the posterior vaginal wall; fundus of uterus not made out. Treatment being unavailing, she went into complete collapse and sank. The intestines were matted together by recent lymph, with dark fluid blood lying between coils of intestine, and a large quantity of fluid blood in Douglas's pouch; uterus in front, not pregnant, containing a little blood and mucus; right Fallopian tube dilated except at opening into uterus, and containing a clot the size of a hen's egg at infundibulum; left tube dilated, inflamed, and contains pus and lymph; both ovaries cystic, the left adherent to pelvic tissues.

I think that, in this case, the sequence of lesions is clear; chronic pyosalpinx of both tubes, inflammation from over-distension of both, hæmosalpinx of right tube, resulting in bleeding from the uterus and

hæmorrhage into the peritoneal cavity, causing pelvic and general peritonitis.¹

This explanation in all probability applies equally to Dr. Simpson's and Dr. D. B. Smith's cases.

A very simple case of *Pelvic Cellulitis* is given in the 'I. M. G.' for October 1871. A Hindoo woman, *æt.* 19, had, about a week or ten days after marriage, a hard painful swelling in the right iliac fossa. The vagina was small, hot, and tender. Diarrhœa was followed by the discharge from the bowels of three ounces of pure pus, disappearance of the swelling and speedy recovery.

Hysteria.

In 1881, the admissions for Hysteria, among the 3,741 soldiers' wives in India, were 22. This being, for the most part, a disease of unmarried life, and there not being a great many single women (excepting Hindoo widows, who are especially secluded) in India, we do not see so much of this neurosis there as we do at home. Still I think that nearly all East Indian women, and not a few of the men, are more or less hysterical. I saw a very painful case of hysterical mania in a remarkably intellectual young unmarried native lady, who, I believe, died unrelieved; and I have for nearly thirty-six years kept in view an East Indian lady, whose case, fully described, would form the ground-work of a volume on the most Protean of all maladies in almost every one of its varieties. Her first attack appears to have occurred in my predecessor's time, when she was over thirty years of age, and the mother of two girls. The immediate cause was the death of a little son. She had a fourth child some years after she had been subject to her malady, the main feature of which was violent convulsions, with apparent unconsciousness and delirious singing. The psychological aspect of her neurosis was very complex. She is a woman of considerable mental power, and an exemplary wife and mother, quite free, in my belief, from all suspicion of intentional feigning. Still, in her person, nearly every known disease was simulated in turn. This was partly explained by the fact that she was one of those whom Theodore Hook designated as 'Buchaneers'—Buchan's 'Domestic Medicine' being, apparently, her daily study. For three years and a half, in a large station, where the duties were almost beyond my power, she gave me an amount of work which would alone have afforded sufficient occupation to a medical man. Her residence and mine were seated on hills between which was a road about a mile long. In one month, I received upwards of *ninety* urgent notes from her very worthy husband, nearly every one of which commenced with such words as,

¹ *Vide* Mr. Lawson Tait's writings—especially proceedings of the Obstetrical Society of London, November 7, 1883.

‘I shall esteem it a favour if you will kindly see Mrs. — as soon as possible ; she has fallen out of bed and broken her back,’ or, ‘she has an attack of lock-jaw.’ I obeyed every one of these calls. The fall certainly appeared to be followed, for a day or two, by weakness of the lower limbs. On the alarm of lock-jaw, I found her mouth so preternaturally widely open, the condyles being as much as possible displaced short of dislocation, that, in any other person, I should certainly have used my thumbs with a view to replacement ; but, considering the great strength of her perfect row of molars—which she used to grind most viciously in her usual attacks—I thought it more prudent to recommend an ether and assafoetida enema, which at once ‘unlocked’ the jaw. In the same way, she had peritonitis, spitting dark blood (real), and a great variety of morbid *simulacra*, among which retention of urine was one of the most troublesome, especially to myself. Having passed the catheter, and completely emptied the bladder of a large quantity of limpid urine, I would be careful to leave without a moment’s delay, and to run down the hill. This could not have occupied three minutes ; when I reached the foot of the ascent, her husband would appear at the top, exclaiming that his wife was not relieved ; I would then retrace my steps and find that, in six minutes, at the utmost, the bladder was quite as distended as it had previously been. When this annoyance had gone on for a considerable time, I recollected that my predecessor had told me that, in a similar attack, he had effected a cure by declaring positively that either her husband or the native sub-assistant surgeon must, in future, pass the catheter. Being about to leave the station, I said that it was absolutely needful that she should become independent of my aid. There were no more attacks of retention.

PUERPERAL DISEASES.

Many of my remarks on the diseases of women combine to show that the English obstetrician, however well-taught and experienced he may be in home work, has many important points to master and facts to learn in Indian practice. Thus the conditions of life and the effects of climate tend considerably to modify the state of the European female during pregnancy and in delivery ; while premature marriage and the usages of her lying-in chamber place the native woman in a position which it takes most of us years of local experience to comprehend fully. Thus all native women and many East Indians maintain the dorsal position in delivery, the midwife squatting in front. The planked *tuckta*, larger than the Bed of Ware, on which the woman lies, the binder, the *goodries* (quilted cotton napkins) are all foreign and strange.

The cord is dressed with a heap of tinder, and, before the unpractised doctor can look round, the infant is dosed with *hot-drawn* castor oil. The new-comer has to become acquainted with scores of quaint and often embarrassing details before he can feel that he is master of the situation in the majority of Indian lying-in chambers.

Intelligent attendance is so frequently absent from the room of an English lady in India, that the medical man will soon find that very nearly all that has to be done, including the washing of the child, will fall to his own hand. Hence the necessity for strict and full preliminary arrangements: so that, as far as possible, everything needful may stand ready, and that nothing of consequence will have to be called for.

Morning Sickness is a frequent cause of suffering and even of danger, when complicated with sea-sickness, on the voyage between England and India. In my remarks on Dysentery (p. 154), I have spoken of the sad complication of that disease with morning-sickness. My friend Dr. Herbert Baillie gave early recognition to the value of the hypodermic injection of morphia in the vomiting of pregnancy.¹ He prescribed that a grain of the acetate be dissolved in half a drachm of water, of which ten minims are injected at the epigastric region, and repeated after one, two, or three hours' interval, till relief occurs. Sometimes one injection proves sufficient, but generally two or three, or it may be even double doses ('i.e. one-third' [*two-thirds*] 'of a grain') are required.

Depraved and Capricious Appetite in Pregnancy.—As I have mentioned at p. 560, the native women of Bengal are much in the habit, when pregnant, of eating portions of thin baked clay cups which are made and sold in the bazars for this purpose. One of my pupils brought me some of these. I thought a morsel which I tasted rather nice. It had the flavour of slate pencil, which I have always liked. At a meeting of the Calcutta Medical Society in July, 1880, Baboo Abinash Chunder Sen, in bringing forward the history of a case of intestinal concretion, mentioned that the woman was said to have been in the habit of eating baked clay biscuits, a practice common among native women in those parts when pregnant.

There appear to be grounds for the belief that natives have or consider that they have means of preventing fecundation. It is stated that Assamese women do not bear children to Europeans; but I have no inclination to sift such trivial information as I have on this disagreeable subject.²

Fifty years ago, when my excellent and very talented chief, Dr.

¹ *I. M. G.*, vol. iv. p. 184.

² There is a curious note on this subject at p. 208 of Webb's *Pathologia Indica*.

Spilsbury, was in his prime, mercury was an Indian fetish at the shrine of which such offerings as the following were laid ¹ :—

‘ Who is not aware that, in this country, many a fond wife has had her wishes crowned in due time after a salivation ? I knew one lady who never bore a child but at the expense of her mouth, and this not once but four times, when death stopped any further trials.’

I know of only one instance of *Triples* borne by a native woman. None of the midwives with whom Henry Marshall conversed ² had ever heard of triplets.

Abortion.—In the long voyage round the Cape, a very large proportion of the English women who were pregnant when they embarked aborted. Now, much of this danger is avoidable, as extremely bad weather is comparatively rarely encountered between Bombay and the Mediterranean ports. I know several instances in which the tendency to this accident became so confirmed that the poor women’s Indian life might be regarded as entirely occupied by a continuous series of abortions. Thirty-six cases of abortion, six of which were fatal, occurred (or rather were registered) among the 4,134 European soldiers’ wives in India in 1880. Ninety-five cases (six fatal) were recorded in the following year, the strength being 3,741. In 1882, there were ninety-one cases, with two deaths, among 3,539 women. In 1883 there were eighty-one abortions with one death. Among the 1,427 cases of delivery reported by Dr. Branfoot at the Madras Lying-in Hospital in 1882, forty-seven were abortions, four of which were fatal. In these deliveries, the duration of pregnancy was calculated to be less than twenty-eight weeks, and the weight of the child less than seventy-eight tolas, or two pounds. In 1883, there were sixty-eight abortions in 1,570 deliveries. Among Dr. D. R. Thomson’s 477 cases of delivery at the Madras Ramasawmy Hospital during that year, there were eleven of abortion. In 1883, there were nine in 442 deliveries. Of the first 244 labours which took place in the Calcutta Female Hospital, thirty-one were premature.³

The wretched history of *Criminal Abortion* in India is given so fully in my work on ‘ Indian Medical Jurisprudence,’ that I need not repeat it at any considerable length here. I have alluded (at p. 417) to the fact that,—in many cases in which we find tetanus, fever, peritonitis, &c. in native women, especially in Hindoo widows, and where the assigned cause of the attack is ‘ cold bathing during menstruation,’ which no Hindoo woman ever does, and which, I believe, is not done by any native or Eurasian woman,—the real cause of disease and death is criminal abortion by the most atrocious means, such as the introduction

¹ *I. M. J.*, p. 160.

² *Notes on the Medical Topography of the Interior of Ceylon*, p. 47.

³ Webb’s *Pathologia Indica*, p. 334.

of a stick of *lal chitra* (plumbago-rosea), a worse irritant than a red hot iron skewer would be, as that would cool, which the virulently acrid stem never does.

The following abstracts of cases, narrated by Dr. Vincent Richards as occurring in the Bancoorah district, were published in the 'I. M. G.' subsequently to the appearance of my work.

In the body of a Hindoo widow 'the intestines were inflamed, the uterus was large, and a thin stick, probably the mid-rib of a cocoa-palm leaf, was protruding through the posterior wall of the uterus, which contained clotted blood only.

In another case, the lower part of the surface of the abdomen and inner side of the left thigh appeared to have been vesicated by the application of some acrid substance, doubtless a stick of *lal chitra*. At the entrance of the vagina were particles of bloody matter, and in the vagina was some dark grumous-looking fluid. On the posterior surface of the vagina, about half an inch from the neck of the uterus, was an opening large enough to allow a finger to pass directly into the cavity of the abdomen. Another wound was found penetrating about one-eighth of an inch into the substance of the uterus, just above its mouth.

In another case of abortion, the foetus and the 'instrument' with which the abortion was said to have been procured were sent to Dr. Richards. The 'instrument' was a twig of *lal chitra* or *lal teetia*, as it is called in that district, about eight inches long. It had evidently been kept for a long time, as it was perfectly dry, and was smeared over, as usual, with assafoetida, but had first been wrapped round with cotton. He understood that several of these twigs are kept on hand by the daees who procure abortion. The woman, a widow, confessed that the man with whom she cohabited went to another village and engaged a professional daee (a woman of low caste, Bagdi), and that the daee procured abortion by means of the twig which she brought with her.

In examining the body of Junghi Bustobi, aged 16 years, who was married, but living as a prostitute with her mother, the abdomen was found covered with *huldee* (turmeric). The peritoneum was intensely inflamed, and a large quantity of pus surrounded the uterus; the bowels were agglutinated together, and the uterus was adherent to the bladder and rectum. Projecting from the left of the fundus uteri was about half an inch of a twig (the material of which it consisted was not mentioned), of the thickness of a crow-quill, and there was an ulcer round it. The os uteri freely admitted a finger. The uterus was large and heavy, and a dirty blackish brown fluid lined it. There was a serous discharge from the vagina.

Prep. F 37, in the Grant M. C. M., Bombay, is 'a uterus at an early period of pregnancy, through which a thin piece of stick has been

passed, piercing in the middle of the fundus, and subsequently the intestine. The attempt to procure abortion caused death on the tenth day.' This much resembles other Bombay cases of the kind given at p. 741 of my 'Indian Medical Jurisprudence.'

Assistant-Surgeon Ram Kishen reported¹ a case of abortion which he had recently observed. The woman had first been given a decoction of one chittack (2 ozs.) of carrot-seeds with two of *goor* (old treacle, which is often a constituent in these abominable compounds) in half a pint of water internally, while a piece of cotton, macerated in honey, on which the following powder was placed, was applied on or within the os: Bindál (*Luffa amara*), zingar (copper rust), aloes, and mamari minak (a compound salt containing chloride of sodium and some hard salt) in equal parts, powdered and mixed thoroughly. It was said that labour pains set in in half an hour, and the woman was delivered in about two hours. He adds that cotton seeds are used to cause abortion.

In one of my last Calcutta University examinations for the Licentiate, I put the question—'What are the native modes of procuring abortion, and what would you say if requested to cause abortion?' The replies generally were very curious.

Deaths in Childbed in India.

I am indebted to my friend the Rev. James Long for a note, from the India Office Records, to this effect: 'In 1703, a surgeon received two shillings and sixpence for each woman delivered alive at any of the Company's settlements, as an encouragement for their [his] "extraordinary" care of such.'

I do not find, in the returns, any notice of the numbers of European soldiers' wives confined. In those of the four years ending 1883, I find the following deaths in the puerperal state (excluding those from abortion): 11, 11, 7, 10. The deaths in childbirth of European soldiers' wives in all India during the four years 1880-83 are given as 9, 6, 1, and 5 in a strength averaging 3,804.

Mr. Hugh Macpherson showed that, during the four years ending 1853-54, the number of soldiers' wives in the Bengal Presidency treated for 'complaints peculiar to women' was 785, of whom 33, or 4·20 per cent., died. From the returns up to 1881, Ewart gives the death-rate of European soldiers' wives throughout India, from diseases peculiar to women, as 3·39 *per mille*. In 1,427 confinements at the Madras Lying-in Hospital in 1882, there occurred 42 deaths, or 2·94 per cent.² Of 477 deliveries in the Ramasawmy Lying-in Hospital, in

¹ *I. M. G.* for April, 1884.

² Dr. Branfoot excludes three cases from his calculation, which gives a percentage of 2·73.

the same year, there were 14 deaths, or 3·13 per cent. Dr. Duncan Stewart's table of 500 cases of labour in the Calcutta Medical College Hospital in 1848-54, gives 34 deaths, or 6·8 per cent.

Holmes's 'Bengal Obituary' contains, in full detail, the epitaphs upon the tombs of 5,901 persons buried in the graveyards of Western India, between the years 1700 and 1848. I find that, of these, 808 were married women who died within the child-bearing ages of 14 and 50. Of these females, 620 died between the ages of 14 and 30, and it is clearly shown that 37, or 5·9 per cent., of these, died within a month of the date of their confinement. The number of those who died between the ages of 30 and 50 was 188, of whom 12, or 6·3 per cent., died within the lying-in month. In 2 cases, the women's ages are not given. Including these, 51 of the total of 808 women, or 6·3 per cent., are clearly shown to have died in childbed. It is, however, almost certain that the deaths of many more than these 51 were due to puerperal causes; still, the figures given above are sufficiently formidable, as showing a mortality of upwards of 6 per cent., not among parturient women, but among married women during the child-bearing term of life. It is clear that all of the 808 women in Calcutta whose death-rates I have given above, either occupied high social positions or were in good circumstances. There, as elsewhere, very poor women rest without monumental inscriptions. It will be borne in mind that McClintock¹ calculated that, in the upper and middle classes alone, one parturient woman in 146 dies in childbed, and that the probable death-rate, in puerperal women of all classes, is 1 per cent.

Last year, Dr. Matthews Duncan remarked that Farre nearly accepted the received view that about 1 in 120 die in childbed or within a few weeks after delivery. Dr. Champneys stated, on the same occasion,² that the total mortality, from all causes, in the General Lying-in Hospital in the last four and a half years, since Dr. Williams and he had held charge of it, was 9 in 1,360, equivalent to 1 in 151 or 0·66 per cent. The last two deaths were in-patients admitted moribund from phthisis. It has lately been stated that the mortality in the Gebärhaus at Prague is 'a trifle above one quarter per cent.'³

I have already shown⁴ that, in Calcutta, in Lind's time, child-bearing was peculiarly fatal; and that, in 1770, Clarke noticed that putrid puerperal fever was extremely common in Bengal. It is just noteworthy that, of the thirteen ladies, of child-bearing ages, whose tombstones of last century are preserved in the old cathedral yard of Calcutta, four died in childbed. Extending only as they do up to the year 1848, the above Indian statistics may be regarded as covering the period of midwife ascendancy in India. Writing in 1837, at the large

¹ As cited by Playfair.

² *M. T.*, vol. ii. for 1885, p. 93.

³ *M. T.*, p. 794.

⁴ Under the heading 'Puerperal Fever,' p. 271.

military station, Muttra, at which there were doubtless many young wives of officers resident, the late Mr. James Macrae said : ‘ The abdomen, after expulsion of the fœtus, is well rubbed down for the purpose of expelling what they call the foul contents of the uterus ; and, on the sixth day, the poor unfortunate woman is subjected to a cold bath, to complete her purification. This mode of practice is the more to be lamented, as it is that to which most European ladies in this country are condemned to submit. Were they aware of the ignorance of the native midwives, they would never employ them ; and even the European women usually employed as midwives are just as ignorant as the natives. In Europe, they generally get some sort of an education ; but, in this country, they receive no kind of instruction ; and it is not to be wondered at if we frequently hear of stillborn children, of ladies dying from uterine hæmorrhage, abdominal inflammation, or *inversio uteri*.’ About the year 1850, I attended nearly all the ladies at a large civil station, but I did not feel much surprised when the magistrate sent for a Calcutta midwife (at an expense of at least 70*l.*) to attend his wife, whom I did not see until she recovered ; and, two years later, I was professionally associated with a lady who made the woman blow into a quart bottle, to ‘ *help the pains* ; ’ and who gave the baby a strong dose of opium. My own experience of disease in Bengal, commencing in 1848, just when ‘ Holmes’s Obituary ’ closed, only enables me to recall the cases of four English ladies who died in childbed, in my practice and within my knowledge. I attended two of these ladies in consultation. The causes of death were, respectively, pulmonary embolism, placenta prævia, and post-partum hæmorrhage (two). In a table showing the causes of death of 640 European Protestant females who died in Calcutta, 1856–60,¹ my friend Surgeon-General Hugh Macpherson has given the deaths in childbed as twenty in number, or 3·12 per cent. of all the female deaths. He adds that the ratio for all England and Wales, as taken from the Registrar-General’s returns, was, on an average of years, about 1½ per cent. of the total female deaths. He argued that the difference was not in reality so much in favour of England, considering how few unmarried and old European women there are in Calcutta, and therefore how much larger a proportion of the total female population child-bearing women constitute there than at home.

From the Bombay mortuary returns for the five years ending 1860, he found that the proportion of deaths in childbed to the total female deaths was as follows, in different classes of the population of that town :

In Europeans		5 per 100.
Indo-Europeans		5·21 „

¹ In his valuable monograph on ‘ The Mortality of Calcutta during the twenty years ending December 31, 1860 ’ *I. A.*, No. 15, p. 79.

Jews	2·4 per 100.
Native Christians	1·92 „
Parsees	1·87 „
Mussulmans	1·84 „
Brahmins	4·29 „

If, therefore, the ratio was high in Calcutta among Europeans, it was still higher in Bombay; and it appeared that Jewesses, no doubt as being themselves Orientals, suffered much less than Europeans. The wives of Native Christians, Parsees, and Mussulmans showed a mortality only a little higher than the English rate; and it was, he urged, reasonable to suppose that, could they command equally skilled attendance, the casualties would be actually fewer than in Europe. Why the mortality should be so much higher in the families of Brahmins than in other classes of natives, he was unable to guess, unless it is attributable to their greater unwillingness to resort to hospitals or to European medical aid in any shape.

Additions to our modern statistics of deaths in childbed of women of all races and classes in India would be most valuable.

Although menstruation frequently occurs very early among girls of all races in India, maturity is often arrived at there, as elsewhere, very much later. Analysis of the 808 Calcutta deaths given above, forcibly illustrates the evil results of the marriage of immature girls, which has always been too prevalent among all classes in India. Thus we find 249 deaths of young women, twenty-four of which were in childbed, up to the age of twenty-five years; while ninety-five of the deaths—six in childbed—occurred at and below the age of 20. In many of the deaths at later ages, the dangers of premature maternity were doubtless operative; we frequently meet with epitaphs on wives of twenty-one or twenty-two who had been the mothers of four or five children. The three earliest cases of death in childbed were at fourteen, sixteen, and seventeen years. I should mention that there is generally no reason to doubt the accuracy of the ages given on Indian tombstones, it having been very customary, from the latter part of last century, to state the age thus: ‘21 years, 10 months, and 10 days.’

The census returns for Bengal, taken in 1881,¹ show that the majority of native girls marry before the age of 15, and that almost the whole of the female population becomes married before the age of 20. Cohabitation does not always immediately follow the marriage of immature girls; but I have shown, in my ‘Medical Jurisprudence,’ that even their position is liable to horrible disaster, and every Indian medical man has more or less lamentable experience of the danger and sufferings of native child-mothers.

Allan Webb cites ² Aldrovandus for cases occurring in Europe, of

¹ As cited in the *I. M. G.*

² *Pathologia Indica*, p. 261.

maternity at eight and nine years. Out of 127 cases of Bengalis reported to Webb, one birth is stated at eight and one at nine.

Mr. Charles Macnamara mentions¹ the case of a Hindoo girl who became a mother at the age of ten years and six months. The medical attendant, Baboo Mokada Churn Sen, said that labour only lasted about six hours, and was by no means difficult. There were living in that house five generations—the father and mother of this infant, their parents, and the mother and grandmother of those latter. The Baboo has often known instances in which Hindoo females have had children at twelve and thirteen years of age, but never one so young as the above.

I was told by a wet-nurse—a very robust Mohammedan, verging upon middle age—that she had her first child at 12—and that this daughter also had her first child at twelve. In the ‘New York Medical Record’ for January 16, 1871, p. 516, it appeared that Dr. Abbot knew of a case in which the mother was 11 years old and the father 16. I note, in my ‘Medical Jurisprudence,’ that the late Dr. Chuckerbutty told me that he was acquainted with an unquestionable case (he knew the mother from infancy) in which a girl became a mother at *ten*. He said that native girls are frequently mothers at twelve and thirteen.

The following case² represents the very frequent termination of such early pregnancies: ‘No 10, a native, age 12, had been in labour several days; all effectual powers seemed to be paralysed when I visited her; the head tightly impacted in a small pelvis. I performed craniotomy. Failing to fix the hook, so as to gain a sufficient purchase for traction, I left the delivery to the natural efforts, the size of the head having been diminished. Death followed in twenty-four hours.’

Mr. Green, who had large experience in these cases, observed: ‘The greatest amount of peril seems to occur in the first labours of very young women. The parts are rigid, the pelvis small, the muscular power and vital energies deficient, the nervous susceptibility readily impressible by any violent disturbance, and this, through lesion of the brain, leading to convulsions, and in many instances the delivery of the woman has not put a stop to the convulsions, which have gone on to a fatal issue. The majority of the children in these cases, when born un mutilated, have been stillborn, the result, probably, of the long-continued pressure to which they have been subject in their passage through the pelvis of the mother. In many of these cases, had they come earlier under judicious management, the result would have been very different, the manual or instrumental assistance would have been afforded in reasonable time so as to have averted the danger of exhaustion of the sustaining power, to have remedied any malposition in

¹ *I. M. G.*, vol. viii. p. 295.

² Mr. W. A. Green, *I. A.*, No. 11, p. 62.

the descent of the child, to have prevented impaction in the straitened pelvis, and to have insured the employment of instrumental measures at the proper time.'¹

As a general rule, after the first, the deliveries of young and healthy women in India are by no means slow or difficult. When I first went to Calcutta, the midwives who were there—for the most part ignorant women—used, I am certain, to calculate upon this fact, not sending for the medical man until almost immediate birth was certain. A tall, pallid European woman in Calcutta was found sitting on the commode, quite unaware that her infant lay suffocating in the liquor amnii with the placenta above it. At p. 766 of my 'Medical Jurisprudence for India,' I have cited other cases of almost unconscious delivery. Dr. George Smith has recorded² the following very remarkable case. A lady was daily expecting to be confined of her second child. Her first had been born, with the usual pains, after a labour extending over six hours. During the night preceding her delivery, she had felt quite well. She rose several times to attend to her sick child. At about 5.30 A.M., she walked from her house to a bungalow where, lying down upon a cot, she experienced a very slight sensation, as if her bowels were about to be relieved; a feeling as if something had touched her body followed, and caused her to ask the ayah to lift the bed-clothes, when, to the surprise and alarm of both, the child was seen perfectly extruded. Dr. Smith found the placenta within the vagina. Mrs. ——— was awake, and yet so little was her notice attracted by her feelings, that the delivery took place unconsciously. Dr. Smith was satisfied that, had she been standing or seated on the night-chair, the result would probably have been instantly fatal to the child. Had she been asleep, the child would have been suffocated. The child, a female, was a little, but not much, undersized. Dr. Smith had most implicit confidence in the patient's statement, having made searching inquiry into the case. About thirty-two years ago, I attended a very healthy young lady who is, I believe, now living, born in India of European parents. She was delivered while standing, so unexpectedly and rapidly, of her first child, at the eighth month, that the skull sustained a fatal fracture by falling upon a marble floor.

Cases of lingering labour from uterine inertia, &c. in anæmiated women of impaired muscular power frequently occur among all races

¹ Doggrel as it is, the following epitaph, which used to exist near Azimghur, gives the history of thousands of Indian cases:—

TO THE MEMORY OF MRS. S. A. AND HER STILLBORN SON.

'Just fifteen years she was a maid,
And scarce eleven months a wife,
Four days and nights in labour laid,
Brought forth, and then gave up her life.'

Holmes's Bengal Obituary, p. 379.

² *I. A.*, No. 8, p. 629.

and classes in India. I was careful to give these women good support, was patient and never used instruments. As far as I saw, the result was always favourable; but, doubtless, thousands of poor native women, living beyond all aid except that of their own midwives, die every year in powerless labour when child-bearing, malarial cachexia, over-work, want, and scorbutus have broken their constitutions.

Here the timely use of instrumental aid in skilled hands proves an inestimable good. I would advise every young medical officer to study Mr. W. A. Green's fifty-two cases concisely related in No. 11. p. 57 of the 'I. A.' It will be seen that, although he could not save those whom he found moribund, he did great good in many otherwise hopeless cases. Few civil surgeons will have so large an experience of such cases as his was in the city of Dacca, where the humanity and skill of such men as Dr. Taylor and himself overcame, to a singular extent, native prejudice against male obstetricians; but all may expect to see more or less of such practice.

The dangers, as from rigidity, &c. which attend the first confinements of women married late in life, have not often to be encountered in India.

To the non-resistance of the maternal structures, so frequent both among Europeans and natives, are probably due the cases of *Spontaneous Evolution* which have been observed in India. The late Dr. Allan Webb reported three cases¹ which occurred under his care while he was garrison assistant-surgeon of Fort William, Calcutta, all in European women. He cites the remark of Burns, who said that, in Glasgow [?], 'which contains not less than 110,000 inhabitants, I cannot learn that more than *one case* of spontaneous evolution has taken place.' In two of Webb's cases, and in one of a native woman seen by Dr. J. A. Green,² the spontaneous evolution of Denman, an 'extremely rare occurrence,'³ was effected, the presenting arm receding and the trunk coming down. In the other Calcutta case, the wife of a medical man, the spontaneous expulsion of Douglas occurred, the head coming down. All of these mothers did well.

Baboo C. K. Mitra has reported the case of a Mohammedan primipara, æt. 20, nearly at her full term, who was spontaneously delivered of a stillborn child weighing 2 lbs. 10½ ozs. The arm had passed the vulva upwards of twelve hours previously. Two native daees had pulled at and broken the humerus; a portion of the funis was prolapsed. The arm came down, and then the upper part of the thorax and the head were expelled in the reporter's presence. 'The neck was curiously elongated.'⁴

Dr. Peter Cullen⁵ attended a native woman, æt. 30, in her sixth confinement. The arm presented. The head, which had been in the

¹ *I. M. J.*, vol. vii. N.S. p. 731.

² *I. M. G.*, vol. i. p. 115.

³ Dr. Lloyd Roberts, second edition, p. 100.

⁴ *I. M. G.*, vol. iv. p. 164.

⁵ *Ibid.* for 1872, p. 108.

left iliac fossa, appeared to become doubled on the chest, the back of the neck and shoulder being the presenting parts. The child was dead ; the mother made a good recovery.

In the Lying-in Hospital, Madras, in 1882, one case (arm and funis) of spontaneous evolution occurred in 1,427 cases of labour. A case (head and arm) also occurred in the same year among the 477 deliveries at the Madras Ramasawmy Hospital. In 1883, two cases (preternatural arm) occurred at the first, and one (transverse arm) at the second of these institutions.

The native treatment of parturient women, barbarous and harmful as it is, resembles, in its leading features, the system of ' *confinement* ' and ' *lying-in* ' which our professional predecessors of the Middle Ages probably learnt in the Crusades. The following statement was made by Modoosoodun Goopto.¹ In Calcutta, 'the woman, after delivery, is placed in a small damp room, very ill-ventilated, with one small door only, no window or opening in the nature of a chimney. The door is always closed. The room is in a corner of the compound. From the moment after delivery, wood fires are kindled in different parts of the room, sometimes two, sometimes three. The smoke is allowed to find its way through the walls and roof. The room is kept at a great heat ; I think it cannot be below 90° Fahr.' [Prosonocomar Tagore said that it was a great deal more.] 'The room is a temporary hut of mats and bamboo, thatched with straw or grass, detached from the house (the woman at this period being considered impure).' 'All but Brahminee women remain impure for a month, the high-caste women for 21 days. P. C. Tagore said, eleven days only.' This hut was generally kept for the purpose of the women of the family being confined in it. 'During the first three days, the woman is given a powder made of stimulating spices, as black pepper, long pepper, and dry ginger. After three days, the same made into a paste with hot water and boiled down.' 'Three or four women out of twenty die of fever and tetanus, produced by this treatment, in six, eight, or ten days after parturition.' This mode of ' *confinement* ' is doubtless not so general now as it was formerly ; but I witnessed it, in a Calcutta palace, about ten years ago.

Second only in misery to the sufferings of the patient are, not unfrequently, those of the accoucheur. My friend, Dr. Herbert Baillie, wrote as follows, in describing a case of arm presentation, in which he delivered a dead child by podalic version. 'The lying-in chamber, if such it may be termed (although actually prepared for the event), was a mud-constructed cabin, (scarce 8 feet by 6, the patient lying on a mat on the ground, as there was no bedstead or means of raising her, save by rolling some clothes into a pillow form and placing them

¹ *Fever Hospital Report*, and Webb's *Pathologia Indica*, p. 336.

under the pelvis). . . . Having cast off my coat, I proceeded to introduce my right hand. . . . lying at full length on the ground made the operation anything but facile, with the aggravation that the hut was full of smoke and that, for the greater part of twenty-four hours, my every movement was watched by hags who never tired.' This was almost precisely my own condition in a case where I performed craniotomy in a remote native hut.

Mootoosawmy Modeliar, one of the first medical graduates in the Madras Presidency who paid special attention to midwifery, describes a high-caste woman as lying in labour 'in a close room, stretched on a mat on the floor, with a block of wood for her pillow.' 'The woman, immediately after confinement, is made to stand up against the wall, the abdomen is besmeared, more especially the uterine region, with oil, friction is used with the palm of the hand, or by the head of the midwife; after which the abdomen is tightly bound with a long piece of cloth twisted.'

For a very recent account of the native modes of treating lying-in women, see 'I. M. G.' for November, 1885.

The following are extracts from a description of Burmese midwifery practice: ¹ When the labour pains come on, the woman is attended by one or two *Woon-zwè* (midwives), and her female friends. All doors and windows are shut, to render the room as hot and close as possible. She is perfectly nude, and, being urged to do so, runs round the room as long as she is able, with or without assistance, sometimes stopping and pressing her loins against the posts, at others raising a heavy weight with both hands and forcibly bringing it down, as if pounding paddy (rice), then falling and rolling on the floor. All this time she is uttering piercing cries, vowing separation from her husband, who sits in the next room or in the street, laughing on hearing himself abused, or, if he be a man of feeling, lifting up the lid of every box in the house as a preventive against charms; or he prepares charmed water, which he sends in to his spouse to drink. At last she falls exhausted on the floor. The women press the child down with their hands, trying to expel it forcibly. Sometimes the midwife sits upon her, or presses the child down with her foot. To bring away the placenta, the abdomen is pressed, the cord pulled, the woman's loins beaten with a hard pillow, or her hair is forced down her throat to cause vomiting. After delivery, four friends lift her up by the hands and feet, bathe her in warm water, and place her as close to a large fire as possible, smearing her body with turmeric mixed with a little *chunam* (lime), and making her swallow a mixture of salt, turmeric, and *chunam*. A hot brick and salt enveloped in a cloth are also pressed against different parts of the body, and often a handful of warm salt is applied and *even introduced*. She is then subjected to *mi-ken* (exposure to fire) for seven

¹ I. M. J., vol. ii. p. 340.

days, during which she is obliged to take the above-mentioned dose three times a day, in order to keep the inside of the body as hot as the outside, and to drink warm water. Once or twice a day she has a kind of vapour bath, and is often made to sit upon a hot brick covered with cloth. Day and night she lies on a plank or bamboo frame only a cubit wide, placed as close to the fire as possible, so that she can only just turn her back or stomach to the heat as she finds it too great on either side. The heat would be intolerable, but that she is every now and then smeared all over with pounded turmeric and water. She is kept in a state of profuse perspiration, from which she is gradually relieved on the seventh day. During these days, a lady of rank was known to have burnt 1,100 large billets of fire-wood, but the usual allowance is 2 or 300 billets. The wood of the tamarind tree is used by those who can afford it, as it is said to make the hottest fire. After this roasting, the skin is quite blackened and peels off.

My friend Dr. Waring's account¹ fully confirms the above description of the Burmese confinement (literally, in their language, 'roasting') in all essential particulars.

As I have already mentioned, natives and many Eurasian women, maintain the dorsal position, as in lithotomy, during parturition. I objected strongly to this when I first went to India, but it enables the *dace* to squat close to the woman, and to use both her hands, and occasionally her feet. Mootoosawmy Modeliar says that, in Southern India, 'A woman in labour is delivered in any position, either on her back or in a sitting posture, and, at the same time, is made to strain, with all her might, in order to assist delivery.'² 'In cases where the foetus is impacted or obstructed from causes unknown, and the midwife is unable to deliver, the woman is placed on her back with legs flexed and abducted, the pelvis fixed by the midwife's feet placed on the groins of the patient, the head of the foetus is besmeared with oil, and, at the same time, the miserable creature is made to strain as much as possible.'

'If the child is supposed to be dead, she perforates the head by means of an iron style curved into a hook. Should the head give way in the extraction, the superior extremities are then detached, one by one, by a small knife, of any description, generally the first that can be had, after which the body is extracted by the hand.'

¹ *I. A.*, No. 1, p. 95.

² When the first Europeans came to Calcutta to lay down the railway, I was called to see one of their wives in labour, and, as I have mentioned above, found her blowing with all her might into an empty quart bottle, a practice which one of her companions, who professed to be a cunning woman, said would 'help the pains.' The degree of civilisation prevailing among the pioneers of plate-laying in India may be gauged by the fact that, at one of the first dinners to which this poor woman 'sat up,' her husband beat her over the head with a hot shoulder of mutton.

An interesting account of the native practice of midwifery in Rajpootana, is given by Surgeon-General Moore,¹ and a chapter in Wise's 'Commentary on the Hindu System of Medicine,' Book V., is devoted to Midwifery and the Diseases of Women and Children.

The amount of mischief done by native midwives (*daees*), and, in old times, by ignorant European women, is incalculable. My first difficult case in India was that of a poor girl living in a hut several miles out of Chittagong. The child had its chin hitched over the pubes, the women in attendance had evidently employed all their force in pulling the body down, and the mother had eclampsia. The maternal parts were dry and swollen, but I succeeded in perforating and delivering. The mother recovered with vesico-vaginal fistula.

Indian medical literature affords abundant evidence of the fact that, where the arm presents, it is the rule, rather than the exception, that the limb should be torn or cut off. Thus we read: 'The child's forearm had been pulled off at the elbow, in forcible attempts to deliver the woman. I delivered by turning, and the woman's life was saved.'² 'The midwife lifted something from under a piece of cloth. It proved to be the left forearm of a child. Delivery was effected by evisceration.'³ The woman ultimately made a favourable recovery.' 'I discovered the left shoulder presenting, and, on turning down the arm, found that it had been wrenched off by the violence employed, a little below the insertion of the deltoid. The midwife had, I imagine, immediately returned the stump.' Still 'the patient made a rapid recovery.'⁴ 'The presenting portion imparted a bony feel to the hand. This was discovered to be the right shoulder from which the arm had been severed by the alternate efforts of five midwives.'⁵ 'The arm had presented, and had been violently torn away at the shoulder-joint by the native nurse.' 'Recovery complete in about ten days.' 'The right hand, which had protruded, had been torn off by a native nurse before my visit.' 'Recovery complete in ten days.' 'Hand and arm first came down; the native nurse cut off the hand.' 'Death on the morning of the sixth day.' 'The hand and arm presented; the limb was torn off violently by the native midwife.' 'Recovery.' 'The arm had presented, and had been violently torn away at the shoulder-joint by the native midwife; the shoulder presenting and much torn.' 'Recovery complete in about ten days.'⁶ It will be noticed that, of these nine cases of avulsion, or hacking off of the arm, one occurred in the Bombay, and three in the Madras Presidency;

¹ I. A. No. 21, p. 109.

² Mr. Leith, *Bom. Med. Phys. Trans.* for 1841.

³ A Cuttack Case, *ibid.*, N. S. iv. p. 448.

⁴ *Madras Quarterly Journal of Medical Science*, &c.—Mr. Tait of Nellore.

⁵ Native surgeon, P. S. M. Moodelly, Tanjore District, *I. M. G.*, vol. ii. No. 3. He reports a second case of this kind.

⁶ Mr. W. A. Green, *op. cit.*

one in Orissa ; and four in Eastern Bengal. Unhappily, native midwives' midwifery varies little throughout the Indian Peninsula.

Barbarous as is this practice of tearing or cutting away the presenting arm, it appears to be quite systematically intentional. M. Modeliar says, in commenting upon the first case of severing the arm cited above : ' I questioned the midwives to ascertain what object, if any, they had in view in conducting the treatment of the case, and learned that, in all cross-births, their plan of treatment is to endeavour to push up the arm and bring the head to the pelvic brim.'

It is a little remarkable, but probably only by coincidence, that in two of Mr. Green's cases where the child had been thus mutilated by the *daees*, the presentation was adjusted by nature, thus :—

' Native woman. The hand and arm presented, torn off violently by the native midwife. Natural birth subsequently took place, the head coming down, the child stillborn ; *recovery*.'

' Native woman, *æt.* 13, first child ; hand and arm first came down, native nurse cut off the hand ; had been in labour twenty-six hours when first visited, pains had then ceased for fourteen hours. It was found that the os uteri was not fully dilated ; at night several doses of ergot and purgative enemata ; on the third day of labour uterine action returned, the woman was naturally delivered by the head. Appeared to be doing well. Puerperal peritonitis on the fifth, death on the sixth day after the commencement of labour ' (p. 68).

Cases of *rupture* and *eversion* of the *uterus*, by *manual violence*, are only too common.¹

Extra-Uterine Pregnancy.

Prep. 939 in the Calcutta Med. College Museum may perhaps be regarded as displaying *Ovarian Pregnancy*. ' The cavity believed to contain an ovum is in the right ovarian region. Its internal wall is rather roughened, and, externally, the sac is matted to the neighbouring parts. The uterus is small, indicating no signs of activity of function.'

Ventral Gestation must have taken place in a lady who came down to Calcutta on her way to England, having, for some time previously, passed foetal bones by the rectum.

¹ Writing on native practice at Muttra, the late Mr. James MacRae (*I. M. J.*, N.S. vol. ii. p. 596) said that the native midwives fix the umbilical cord to the patient's thigh to prevent the placenta from ascending into the stomach. The usual practice is to allow the child to remain attached to the cord until the placenta has come away, however long that may be ; and, when they deem it proper to remove the placenta, they always do so by pulling at the cord. By the first piece of malpractice the child, if weakly, is sure to be lost by cold and exposure, and by the latter, the mother is frequently injured for life, and is obliged to drag out a miserable existence with a prolapsed and inverted uterus protruding at the external parts.

Ventral gestation also occurred in the following case : ¹ Baboo N. C. Mitter examined the body of a low-caste woman of Oraic, sent by the police for inspection. All that was known was that, in an attempt to induce abortion, death ensued. A thin, transparent membranous bag of fluid was found lying across the hypogastric region, inclining more to the right. It contained a fœtus of about the fourth month, separated by a partition of amnion. Two placentaë were attached to the broad ligament and to the soft parts of the pelvic cavity, which was filled with half-coagulated blood. The uterus was enlarged to four times its natural size, but its internal surface showed no signs of conception. The os was soft and dilated to the size of a rupee. The vagina was bloody and was plugged with a ball of rags containing some dark greenish substance, apparently used for the purpose of inducing abortion. The petticoat was considerably blood-stained. There was no breach of continuity in the uterus and Fallopian tubes.

Prep. 107, Sect. 13, in the Madras M. C. Museum represents abdominal extra-uterine gestation in a cachectic Hindu, *æt.* 36, in her fourth pregnancy. It was considered that pregnancy had occurred in a retroflexed uterus. From the time of her admission to the morning on which she died, she continued to suffer daily from spasmodic pain in the abdomen, during which the contractions and relaxations of the uterus seemed to be felt. While she appeared to be sleeping, collapse set in, and she died in about half an hour. A large quantity of blood occupied the left of the abdomen and uterus. There was recent peritonitis. Behind the uterus was a large sac which, with the uterus, occupied the pelvic cavity and lower part of the abdomen. This sac was connected with the lower intestines by firm adhesions. It was attached to the uterus, entirely obscuring all but its front surface. It contained a fœtus as large as a six-months' child. At its back part was a rent through which hæmorrhage had occurred into the abdominal cavity, but not into that of the amnion. The placenta appears to have been attached to the posterior surface of the uterus. That organ was somewhat enlarged, and its cavity was lined by decidua.

Tubal Gestation.—Dr. J. S. Login ² examined the body of a woman who was found dead in the lines of a native regiment. It appeared that she had been exposed to mental anxiety and distress for some time before her death, and had been subject to occasional attacks of severe abdominal pain in the right hypogastric and inguinal regions. During the last three or four days, the paroxysms of pain had been accompanied by attacks of syncope, on the subsidence of which she was able to resume her usual occupations. On the day of her death, these symptoms were said to have occurred with unusual violence. A large coagulum of blood was found to cover the surface of the intestines.

¹ *I. M. G.*, vol. i. p. 90.

² *C. M. T.*, vol. viii. appendix, p. 1.

There was a collapsed sac communicating with the right Fallopian tube, the uterine extremity of which was obstructed. The tunics of the sac adhered anteriorly to a small extent to the abdominal wall. Posteriorly, a fissure, two inches long, exposed to view the ruptured membranes which had contained a foetus of about ten weeks old, which was found among the coagula in the cavity of the pelvis. The uterus was enlarged to the extent usual in the second month of pregnancy and had a deciduous lining.

My friend, Dr. Thomas W. Wilson, tells me of the case, not in his own practice, of the wife of a Bengal civilian, about 27 years of age, pregnant for the first time, in whom rupture of a tubal pregnancy occurred as she was stepping into her carriage. She survived the accident, in a state of collapse, for several hours. Here and in the following case there was time for surgical interference. See Mr. Lawson Tait's 'cases of extra-uterine pregnancy operated on at time of rupture.'¹

Dr. Allan Webb² was called to a married Bengali girl, *æt.* 16, when dying from internal hæmorrhage. It was found that the peritoneal cavity was distended with fluid blood. All the structures had a blanched appearance. The pelvis was filled with a tolerably firm coagulum of black blood. In the right *Fallopian tube was an enlargement about the size of a large walnut* with which the coagulum was intimately connected. 'This enlargement was exceedingly thinned anteriorly, with one or two black spots ready to give way; whilst, superiorly, it had burst and given exit to a small foetus. The placenta had not wholly escaped, part of it being still adherent to the internal surface of the Fallopian tube; the free villous surface seeming to consist of the torn mouths of an immense congeries of small vessels, whence the hæmorrhage had come. Foetus, placenta, and membranes together were not bigger than a good-sized walnut.' The uterus was of the natural size, was closed with tenacious secretion and lined with decidua.

Dr. John Macpherson reported³ that the body of B., dead about twelve hours, alleged to have received a beating from her husband, and to have died soon afterwards of cholera, was brought to him by the police for examination. The body was that of a healthy woman. The abdominal cavity was quite full of blood, chiefly fluid, but mixed with coagula. The whole cavity of the pelvis was occupied by a pretty firm coagulum. The bleeding had been caused by rupture of the left Fallopian tube. No ovum was found. A bristle was passed without difficulty through this tube, for an inch up to the wall of a tumour about the size of a pigeon's egg, whence the hæmorrhage had pro-

¹ *British Medical Journal*, vol. i. for 1884, p. 1250.

² *Pathologia Indica*, p. 294. The preparation is described at p. 289.

³ *Ibid.* p. 323.

ceeded. The coats of the tube forming the walls of the tumour were much attenuated and showed a purple discolouration. There was a small rent in them posteriorly, not large enough to admit of the passage of the point of the little finger. The interior of the tumour was occupied by a firm coagulum; no trace of the recent attachment of the ovum to it was observed. The os tincæ and cervix were firmly agglutinated by the secretion of the Nabothian glands; the body of the uterus was perhaps a trifle enlarged; interior slightly vascular and lined with jelly; no trace of a decidua.

Dr. H. Clark¹ received into his hospital a delicate European woman, *æt.* 26, who had been suddenly seized with pain in the belly and collapse, evidently from internal hæmorrhage. She rallied, and, six days later, was very well and felt inclined to eat. On the previous day, she passed per vaginam a fibrinous clot about the size but not of the shape of the interior of the unimpregnated uterus. Some blood came at about the same time, and continued for two days. On the eleventh day, she 'had a relapse.' On the fourteenth day, she had appeared to be getting on well when acute pain came on suddenly when she was at stool, and a swelling in the right iliac region which had been noticed on the occasion of the first attack, but which, having disappeared that afternoon, had been attributed to muscular spasm, reappeared extending from the anterior superior spine of the ilium to the umbilicus. On the sixteenth day, there was an attack of pain after defæcation, and, late in the same day, she became very faint. There was some fever which yielded to quinine. On the twenty-fifth day, there was pain in the tumour to which leeching gave some relief. She lived sixteen days longer; there were occasional attacks of faintness. There was occasionally a dark discharge from the vagina. The tumour remained, and, latterly, was felt from the rectum. Towards the last, there was tympanitis. Extending some way above the uterus was a large sac which also stretched deeply down between the uterus and rectum. Its capacity was about sixteen ounces. It appears to have been filled with fibrinous clot. To the right of this sac, but with a cavity quite distinct, was an oval body with a rough, red external surface. It contained a fœtus three and a half inches long, with an umbilical cord and placenta. Expanding over and losing itself in the tumour was the right Fallopian tube, which, at its distal extremity, was enlarged so as to allow a director to be passed along it some way. The tube was about three inches long when it thus became lost on the tumour. Between the cavities of the two no continuity could be traced. There is no mention of decidua.

Prep. 51, Series 13, in the Catalogue of the Madras Collection represents tubal extra-uterine gestation. Rupture of the Fallopian tube occurred and caused sudden death. There was suspicion that

¹ *I. A.*, No. 14, p. 494.

death had been caused by a kick, but this is considered to have been disproved by the autopsy. A corpus luteum can be distinctly seen in the corresponding ovary.

Prep. 97 is a uterus and appendages from a case of extra-uterine tubal pregnancy. Death from rupture of the sac, and hæmorrhage. A native *æt.* 17, with no children, was suddenly seized with pain in the lower part of the abdomen when four months pregnant. The pain was followed by symptoms of internal hæmorrhage and shock with which she died in thirty-six hours. A large quantity of blood was found in the abdomen, the tissues of the body were generally exsanguine. The right Fallopian tube was distended to the size of a hen's egg, and a rupture had taken place in its coats. An ovum was in the tube in a vascular sac the size of a walnut, with a small embryo within. The uterus was slightly enlarged and lined by a decidua and with some blood in the cavity.

Prep. 50 is also one of tubal extra-uterine gestation.

Dr. Wilson relates¹ the case of a Hindoo woman *æt.* 21, who was admitted to his ward, pregnant, and in a state of anæmia and general anasarca consequent upon an attack of cholera. She was expected to reach her full time in the first week of July. The child was last ascertained to be alive on May 10. On the 18th she said, 'My child is dead, it has not moved for some days.' On July 3, there commenced a continued dribbling from the rectum of a turbid fluid similar to that which was found to exist in the sac containing the child. Rigor and tympanitis set in and she died on the 6th. The cavity of the uterus was found to be in its natural state. Surmounting the fundus was a sac, without any distinctive membranes, its walls adherent on all sides to the contiguous parts, out of which it seemed formed; it contained a child of apparently eight months' development and a placenta attached to the exterior of the fundus uteri. Whether this was a *Ventral* or an *Interstitial Pregnancy* appears doubtful. In another case of a native woman, *æt.* 23, narrated by Dr. Wilson in the same communication, there was evidently *Interstitial* gestation, a decomposed $5\frac{1}{2}$ or 6 months' fœtus lying encysted in the site of the junction of the Fallopian tube with the uterus.

Preparation 938, in Ewart's Catalogue, although imperfect, represents *Interstitial Extra-uterine Fœtation*. 'It would appear as if nature had set up a process for the admission of the embryo into the uterus, for there is a distinct sac or cavity in the right corner, lined with black coagulum, and the wall of the uterus is as thin here as a shilling; whilst, elsewhere, it is as thick as a finger.' 'The interior surface of the uterus is lined by decidua.'

OBSTRUCTION TO LABOUR BY MONSTERS is by no means of exceptional rarity in India; but, unfortunately, the reports do not always

¹ *I. A.*, No. 5, p. 246.

specify the race or give the manner in which the monster was born. Henry Goodeve's description of Prep. 974 in Ewart's Catalogue, is thus defective. It 'consists of two female children, united together in the thorax and upper part of the abdomen by a broad connection which extends from the sternum to the umbilicus.' 'One thoracic and one abdominal cavity were common to both children, these being divided by a single diaphragm.'

Dr. S. A. Ranking has described¹ the condition of 'Siamese Twins' (Hindoo) three months old.

The objection made above applies to the following cases marked thus *—although I believe that most of them, if not all, were native.

*Prep. 975 Ewart's Catalogue. Twins at the full term connected in the umbilical region in the manner of the Siamese twins.

*981. A double monster with only one face to two heads and two bodies which are joined together along the thorax and abdomen by their anterior aspects.

*Webb's No. 539 was a bicephalous foetus in which there were a double head and neck, a double heart and double stomach, the lower parts being single.

*977. A bicephalous foetus.

*982. A double monster with one head and face and two bodies. Thorax and abdomen are joined together.

Dr. W. P. Harris describes² a monster of this kind born of a poor native mother without difficulty, as it was immature. There was only one brain, but there were two bodies joined together, front to front from above downwards as far as the navel.

*6 of Addenda Miscellanea p. 322 of Ewart's Catalogue. Twins; one is fully formed, the other is acephalous, quite rudimentary, and attached to the epigastrium of the fully-developed child by its abdomen.

Dr. James Cleghorn has given³ a full description of a native male child which, when he saw it, was fully-grown, fairly-developed and vigorous, but which died in twenty-six days. It had, attached to the lower part of its epigastric region, towards the right, a pelvis and a pair of legs which, in appearance, might have belonged to a foetus of the sixth or seventh month.

*976. Twins attached to each other in the umbilical region. One is a full-grown foetus. The other is diminutive in size and malformed.

Nos. 757 and 758 in Webb are models of a Hindoo youth and of a Chinaman in each of whom a twin hangs by the neck from the brother's chest.

*No. 973 in Ewart. A foetus with an enormous hydrocephalic expansion of the bones and cavity of the cranium.

¹ *I. M. G.* for 1877, p. 158.

² *Ibid.* vol. ii. p. 73.

³ *Ibid.* vol. vi. p. 9.

*No. 678 in Webb's Catalogue. 'The neck gave way from the application of the forceps to the body. This was followed by a gush of water, thought to be occasioned by rupture of the bladder. But the collapsed hydrocephalic head, whence the water came through the spinal canal, soon followed.'

Cæsarian Section.

'When a pregnant woman, near the full time, is suddenly killed, as a goat is killed before the altar, and should the child move in the uterus, an incision is to be made in the abdomen, and the infant removed.'¹

The information regarding *Pelvic Deformity*, as it occurs among natives in India, is very small.

Judging from the descriptions given in the Calcutta catalogue, the eight pelves, numbered 14 to 21, at page 322, fully illustrate typical deformity from rickets and malacosteon; but, unfortunately, there is no allusion to *race*.

The following may be given here, as it is one of the only two cases of *rickety* deformity, in a native, which I can discover. Dr. J. Shortt² was called to see Venkumah, *ætat.* 21, basket-maker, at her own quarters. Her height was 3 feet 6 inches, colour black, head large, forehead prominent, with high cheek bones, flat nose and thickened alæ; lips thick and pendulous, trunk short and well-formed, extremities short, thick, and fleshy. When first seen, she had been in labour for four days. The membranes had been ruptured forty hours; the presentation was natural. Fœtal head high up in the pelvis; no fœtal heart-sounds, passages dry, vulva swollen and tumified, great deformity and narrowing of the pelvis in its antero-posterior diameter, admitting only two fingers on the right, and one on the left side. As she was evidently almost exhausted and moribund when first seen, and, as the child was dead, the Cæsarian section was evidently inadmissible. Craniotomy was attempted unsuccessfully from the contracted state of the passage. *P.-M.*: The uterus was found firmly contracted over a female child; fœtal head impacted over the brim of the pelvis; the latter much distorted; bladder ruptured transversely to the extent of 3 inches. Fœtus apparently healthy and well-formed, weighing 7 pounds 2 drachms, and measuring $17\frac{1}{4}$ inches in length.

The second case of rickety deformity occurred to Dr. P. Cullen³ in a strong-looking native primipara, *ætat.* 20. The promontory of the sacrum was much pushed forwards, causing antero-posterior distortion of the pelvis. The child was dead, with the arm and funis presenting. Cæsarian section. The mother died in three days.

¹ Wise, *Hindu System of Medicine*.

² *I. A.*, No. vii. p. 292.

³ *I. M. G.* for 1872, p. 108.

Two Mussulmaunee women, in whom Sir Joseph Fayrer¹ performed Cæsarian section, evidently suffered from *Malacosteon*.

The first, *æt.* 35, was much deformed, being short of stature, and the legs being crooked and bent forwards. The spine was curved laterally and posteriorly. The tubera ischii were closely approximated, and the rami of the pubes were almost in apposition. The outlet was reduced to a passage so small that one finger could with difficulty be introduced. As the only chance for life, the child was removed by the Cæsarian section. It was so monstrously deformed that the female attendants were with difficulty made to give it proper care. It was a male, weighing 8 lbs. Its length was 1 foot 8½ inches; the circumference of its head was 1 foot 8 inches. It lived for about twelve hours, sucked some milk, and passed meconium. The mother sank in forty-two hours. The uterus had contracted, but the wound was not completely closed, blood had escaped and had given rise to peritonitis.

A Mussulmaunee slave, *ætat.* 30. She was not much deformed, the limbs being natural, though the trunk was short and dwarfish. The pelvis was small; the inlet contracted to a diameter of less than three inches each way. It was with difficulty the finger could reach the os uteri, the sacrum bulging forwards, and the tuberosities of the ischia and the rami of the pubes being in close approximation. It was her first conception. She said that the time was considerably over nine months, that she had been in labour for eight days, and that the liquor amnii had been discharged on the previous day. Two dead female children, attached to one placenta, were removed by Cæsarian section. The attachment of the placenta to the anterior surface of the uterus gave rise to great hæmorrhage during the operation. She survived the operation two days, dying apparently under the combined influence of peritonitis, shock, and hæmorrhage. The uterus had contracted so much as almost to close the wound—the os uteri was contracted to a point.

Dr. Tasse, of Chandernagore, performed Cæsarian section successfully.² The report is a mere slovenly paragraph, in which the case of the patient and the character of the deformity are not mentioned. The woman was aged 20 years, had a deformed pelvis, and had been in labour four days. The child was dead. The wound was healed in forty days.

Our Indian medical literature contains several reports of cases of gastrotomy where the women were either moribund or dead.—Death from laceration of brain, Cæsarian operation, spontaneous expulsion of the placenta, in a European, (Allan Webb, 'Pathologia Indica,' p. 308.)

Native, *æt.* 18. Arm presentation, limb torn off, funis torn, vagina and urethra dreadfully torn. Gastrotomy; death in three hours.

¹ *I. A.*, No. 5, p. 89,

² *C. M. T.*, vol. vi. p. 495,

(Dr. P. Cullen, 'I. M. G.' for 1872, p. 108). Gastrotomy *in extremis*, (Mr. Minas, *ibid.*, vol. viii. p. 237).—Labour complex, Rupture of Vagina; Gastrotomy, Transfusion,—and Labour Complex, Ruptured Uterus and Vagina; Gastrotomy. These two are numbered xvi. and xix. in Dr. Branfoot's Report of the Madras Lying-in Hospital for 1882. In the following year, Dr. Branfoot reported two cases of delivery by gastrotomy, No. xxiii. :—Labour Complex, Ruptured Vagina, and No. xxviii., Labour complex, Rupture of Uterus and Vagina. Both cases were fatal.

Rupture of the Uterus

Is undoubtedly, in some cases, caused by mala-praxis. Some years ago, a Calcutta druggist was in the habit of selling a nostrum, the main constituent of which was ergot of rye, as a means of expediting lingering labour. Within my knowledge, a poor native woman died from rupture of the uterus consequent upon taking this stuff.

This man was a quack of the old school. Fifty years ago, ergot was employed as loosely as carbolic acid and chloral were on their first introduction. I have now before me a paper on the use of ergot in protracted labour, by a distinguished authority, which may have prompted this man's destructive practice.

Mr. James White recorded¹ the case of a Burmese woman, *æt.* about 28, who sustained rupture of the uterus, with complete escape of its contents into the abdominal cavity, after having been two days in her seventh labour. The descriptions of the practices of Burmese midwives which I have cited render it likely that this casualty is not very rare in Burmah. Mr. W. A. Gardiner narrates² the case of a native woman, about 27 years of age, who died from rupture of the uterus in her third labour. The child was hydrocephalic. The reporter says: 'The head was lying free in the abdomen, external to the uterus, the rent in which was tightly clasping the neck. I first got the head back into the uterus, by passing my hand high up in it, and forcibly drawing on the lower limbs, my hand several times was rendered quite powerless by the uterine contractions; and, notwithstanding the uterus had been in action over four days and was ruptured, it took almost all my strength, exerted continuously for nearly two hours, to effect delivery.'

Mr. J. F. Clarke reported in the 'Lancet'³ the case of a woman, *æt.* 34, who made a good recovery from an extensive rupture of the uterus which occurred in her fourth labour.

Only one (fatal) case of rupture of the uterus occurred among the soldiers' wives in the four years 1880–83.

In 477 labours at the Ramasawmy Lying-in Hospital at Madras in

¹ I. A., No. 11, p. 177.

² I. M. G., vol. vi. p. 142.

³ Cited in the I. M. G., vol. v. p. 40.

1882, there was one of rupture of the uterus which was fatal. There was also one fatal case there in 1883.

Dr. Branfoot has given an interesting abstract of eight cases of ruptured uterus and vagina which occurred in 1427 confinements in the Government Lying-in Hospital, Madras, in 1882. There were three cases of ruptured uterus and vagina; one of ruptured uterus; three of ruptured vagina; and one of ruptured vagina and bladder. Of the women who sustained this lesion, two were aged 16; one, 28; three, 30; one, 32; and one, 35. With regard to the *pregnancy*, 3 occurred in the first; one in the fourth; 2 in the sixth; 2 in the seventh. One occurred in the twelfth *hour of labour*, and one each in the 16th, 24th, 26th, 27th, 32nd, 48th, and 72nd. The *presenting part* was head 5; breech 1; arm 1; head, hands, and funis 1. The mode of delivery was—decapitation 1; gastrotomy, 2; cephalotripsy, 4. One case died undelivered; and in one of the cases in which gastrotomy was performed, transfusion was resorted to, the woman having lost a great deal of blood before admission. Of the eight cases, 7 died. The case of rupture of the vagina and bladder remained on January 1, 1883, and was in a fair way towards recovery. This case was a solitary instance of recovery from this most fatal accident; but it served as an encouragement for treatment. Dr. Branfoot considers that, in Madras, ruptured vagina is far commoner than ruptured uterus: and that, when rupture of the uterus does occur, it is usually secondary to the vaginal laceration. The vaginal laceration is almost invariably transverse and is probably caused by a strongly acting uterus dragging on a vagina which is tightly fixed by an impacted presentation. Full details of these eight cases are given at pp. 93-4 of the Report. The following case is noteworthy: A Hindoo, *æt.* 30, sixth pregnancy at full term, an out case, admitted apparently moribund, 26 hours in labour. Pulse small (148), temperature normal, hands and feet cold, countenance anxious, restlessness. The bladder apparently contained urine, but, on introducing a catheter, which was done with difficulty, owing to the pressure of the head upon the pubes, a little dark grumous fluid and fetid gas escaped, the catheter having passed through a rent in the neck of the bladder, and passing thence into the uterus. The abdomen was irregular, uterus in a state of spastic rigidity and lying to the left side, slight pains present, foetal heart inaudible, vagina much swollen and bruised, head at the brim with a large scalp tumour, in the fourth position, firmly impacted; liquor amnii decomposed; some dark fluid escaped during examination. She was freely stimulated and delivery was effected by cephalotripsy. Examination now revealed a rent in the vagina across the anterior wall at its junction with the cervix, the cervix hanging loosely in a gangrenous state. The placenta, lying loosely in the uterus, was removed. Some free bleeding occurred, which was controlled by pressure

and ergotine. A gangrenous flap of cervix was ligatured and cut off with scissors. She was ordered æther hypodermically, and two drachms of brandy every half-hour till she rallied, hot-water bottles to extremities, milk and broth as nourishment, and opium as required. The sloughs separated without any very serious constitutional disturbance. In twenty days, she was in a fair way to recovery. There was still a large opening into the bladder from the loss of almost the whole of the anterior vaginal wall.

In Dr. Branfoot's report for 1883, 6 cases are noticed,—of ruptured uterus and vagina 2; ruptured uterus 2; ruptured vagina 2. There were 2 deliveries by version and 2 by gastrotomy—2 died undelivered. Five of the six cases proved fatal. In the case which recovered, a Mohammedan woman *æt.* 35, there was a fibroid tumour of the uterus. After delivery by version (the shoulder presenting) a rupture of the uterus was detected. The tumour sloughed, and was expelled in masses. The woman was removed from hospital on the 12th day, was doing well and refused to remain. The whole fibroid mass appears to have become enucleated, the rent in the uterus had healed, and the organ had undergone considerable involution.

Accidental traumatic rupture of the uterus is not invariably fatal.

A case by Dr. Spilsbury, afterwards head of the Medical Department, which was long known as 'dear old Spils' case of meddlesome midwifery,' appeared in the 'Transactions of the Calcutta Med. and Phys. Society.'¹ The wife of a Madras sepoy, far advanced in pregnancy, was riding on a bullock, when the animal stumbled and fell. She was thrown forward on one of the animal's horns, which entered about an inch above the pubes. Through the wound, which was about two inches long in a horizontal direction, the child's hand protruded with slight hæmorrhage and discharge of liquor amnii. Labour soon commenced on irritation of the os tincæ: the child, which was apparently at its full time, scarcely breathed, having been injured by the horn. Considerable protrusion of the intestines took place (externally), but was easily reduced. At first, there were considerable pain and tenderness above the navel, uterus well contracted and not particularly painful, urgent thirst and quick pulse. These speedily yielded to treatment. For some days, pus was discharged by the vagina, but she appears to have made a good recovery.²

The Dutch fœtus seems to be remarkably tenacious of vitality. Professor Van Kampen, in his work on Holland and Belgium, tells us

¹ Vol. vii. part 2, p. 467.

² We are told that, when Spinola besieged Bergen-op-Zoom, a woman near her time, fetching water, was cut off in the waist by a cannon-ball, and her lower parts fell into the water. People ran immediately to her, and saw a child stir in the water. It was drawn out and carried to Don Cordua's tent, where it was carefully attended. Afterwards it was taken to Antwerp, and the Infanta Isabella caused it to be christened by the name of Albertus Ambrosas.

that the church at Zaandam possesses a picture descriptive of a curious incident. A furious bull had killed his master in the presence of his wife, who was *enceinte*. In an agony of despair, she rushed towards the ungovernable animal, which, with its horns, tossed her into the air. The infant, to which she gave birth in dying, survived the catastrophe.

As the mechanism of labour and the accidents to which it is liable are the same everywhere, and as this is not a treatise on midwifery, I shall only cite here a few noteworthy cases illustrative of peculiarities in Indian practice.

Sub-Asst. Surgeon Bindabun Chatterjee recorded ¹ a case of *Rupture of the Child's Head* in the second stage of labour. A Hindu female, *æt.* 32, delivered in hospital of her second child. The presentation was natural, and the labour not unusually tedious. The child was born dead in consequence of rupture of the scalp and escape of the substance of the brain which occurred during the second stage. The scalp was found much attenuated, and a rupture had taken place posteriorly. The line of rupture was even and horizontal, about the level of the upper part of the occipital bone, and two lines running forwards in a straight direction from the extremity of the horizontal line. The rupture was symmetrical, the lambdoidal suture was also ruptured; child small, and not at full term.

Dr. Branfoot's 35th case, Report of 1882—*Labour Complex, Hepatic Abscess*. An emaciated Hindu woman, *æt.* 30, fifth pregnancy. An out-case, was admitted, October 16, complaining of acute pain in the right axillary region. The respiration was short and painful, 48 per minute; was eight months pregnant. Dulness over the base of the right lung posteriorly, extending about half-way up the chest, and in the right axillary region. Fine crepitation and tubular breathing heard over the same regions, and a friction sound in the lower right axillary region; temperature 101·6, pulse 120. The symptoms subsided somewhat, but pain in the side in a lessened degree, with some tenderness and inability to lie on the right side, continued. The abdomen was pendulous, uterus corresponding to seventh month of gestation, foetal heart audible. She was delivered of a living premature child on the 21st; fever continued, temperature ranging from 100° to 102° F., and pulse 120 to 132, and there was slight diarrhoea. On the 30th, there was a rigor, and bulging of the right side of the chest was found with deficient movement of the chest-wall. On the evening of November 1, the liver was found to be low down in the abdomen, the lower limit being some three inches below the margin of the costal arch. Hepatic abscess with pneumonia of the right lung was diagnosed; but, there being no apparent urgency in the symptoms, it was determined not to tap until the following morning. At 11 P.M., however, she suddenly cried out with pain, and died almost

¹ *I. A.*, No. 3, p. 469.

immediately. An enormous abscess was found in the right lobe of the liver, with peri-hepatitis; the right lung was greatly compressed, the heart was displaced upwards to the left. Sudden death had evidently occurred by an increase in the pus displacing the heart and interfering with its action. No further visceral disease was found.

Labour Complex, Lead Poisoning, Dr. Branfoot's 34th Case, Report for 1882. An East Indian woman, *æt.* 27, in her first pregnancy, admitted on April 25 in the eight month of gestation, suffering from chronic lead-poisoning with extreme anæmia and anasarca and weak heart. The conjunctivæ were slightly tinged yellow, dry hacking cough, swelling of both feet, lips pale, gums spongy and bleeding easily, having a bluish line near junction with the teeth, tongue pallid and fissured, a small circular ulcer under the tongue at the frænum, patches of superficial ulceration at back part of mouth, violent attacks of spasmodic colic pain, particularly felt on right side at hepatic flexure of colon. There was no paralysis or marked wasting of extensor muscles, urine light-coloured and free from albumen. She had been in bad health for some months, had suffered from malarial fever and was in the habit of drinking large quantities of soda-water from a seltzogene, the cover and tap of which were composed of soft lead. The colic was relieved and she improved somewhat in general health. Labour set in on May 10. She was delivered of a putrid male child in eight hours. She became collapsed soon after delivery, but was kept alive for four days by stimulants and nutrient enemata.

Baboo Bindabun Chatterjee has given the following case of *Polypus Uteri, Complicating Labour*: A country-born woman, *æt.* 46, mother of eight healthy children, the last being eight years old, had been in labour for two days. It was found that a large polypus filled the pelvic cavity. It was darkly livid, firm, smooth and glossy. The labia were enormously swollen. The head was pushed back as soon as possible, and the polypus firmly but gently pulled down by polypus-forceps. With some difficulty, a stout cord was passed as high up as possible about the neck of the tumour, and the mass was removed entire by long-bladed curved scissors with blunt points. The head, coming down in about half an hour, was delivered by forceps. A second smaller polypus was found springing from the inside of the os uteri near the root of the other. The ligature came away during delivery, but, as there was no hæmorrhage from the stump, it was left alone. On the fifteenth day, a large slough came away, including, along with the necks of the two polypi, a considerable portion of the cervix, but nothing untoward followed, except a paralytic condition of the bladder, which continued for two months.

Frequent relaxation of the uterine ligaments is at home, those miserable cases of *delivery complicated with prolapse of the uterus* which now and then occur among neglected Indian women must be very rare in Europe.

Dr. Branfoot's 38th Case (1882) is one of *Complex Labour, Prolapse of Uterus, Hypertrophic Elongation of Cervix, Death from Septicæmia*. A Hindu woman, *æt.* 40, admitted when twenty-six hours in labour with her third child at full term, foetal heart audible, the cervix uteri was found protruding from the vulva, œdematous, of an intensely livid colour, and bruised in parts. The os was almost fully dilated, membranes absent, said to have been ruptured when labour set in; head presenting low down in the first position, pains feeble. The cervix was pricked with a needle, and a good deal of serum oozed out, which reduced its size. Pains continuing feeble, and labour not progressing, delivery was effected by forceps; incisions were made all round to prevent laceration of the cervix. A still male child was extracted; the left side of the cervix lacerated up to the vaginal wall, the placenta was expressed thirty minutes after, without hæmorrhage. Four days later, the cervix was gangrenous, fever preceded by rigors occurred; parts of the sphacelated tissue were removed. She died from septicæmia on the tenth day.

The following very remarkable case is recorded by P. S. Mootosawmy Moodelly:¹ Soornam, *æt.* about 30, a tall, delicate-looking woman of the working class, in labour with her eighth child, her youngest being three years old, states that prolapse of the uterus first occurred about three months after the birth of her first child, while she was in the act of lifting a heavy load, and that she has suffered much from the protrusion ever since, particularly in the last month of her pregnancy, when she is obliged to keep her bed from sheer inability to move about. In this state she has given birth to three living children. She says that, the prolapses having increased since her last confinement, she is apprehensive of danger on this occasion. It was learnt, on admission, that the labour pains had set in four days previously; strong at the commencement, they were now weak and recurred at long intervals. The membranes had been ruptured twenty-six hours. Pulse 140, small and quick; tongue thickly coated, moist, thirst urgent, no motion for four days; constant inclination to micturate, but the urine is passed in very small quantities at a time. The bladder was distended, the abdomen hot, the uterine tumour was large, uterus prolapsed and hanging without the vulva; passages hot, but moist, head presenting very low in the sacrum and impacted [this is not a clear description]. The four midwives who accompanied her said that the head had not advanced for eleven hours. An enema produced only a scanty evacuation, the catheter could not be passed. A sedative having been given, the head of the child, which was dead and putrid, was perforated. The cranial bones being strongly ossified, delivery was completed with difficulty by craniotomy-forceps. She became very weak during the operation, and had to be supported by

¹ *I. M. G.*, vol. ii. p. 45.

stimulants &c. After removal of the placenta, the uterus was returned and retained in place by a ball of tow enveloped in silk, secured by a T-bandage. She complained much of after-pains and thirst. On the third day, she had dysentery; the straining caused prolapse of the uterus which was replaced and retained in position by an oval pessary. Under judicious treatment, she was 'quite well' on the sixteenth day after delivery. While in hospital, she was kept in the recumbent position. The judicious reporter gave another case of a wealthy native woman in labour with her fifth child. Uterine prolapse occurred in her second confinement, since which her general health had been bad. She was weak and emaciated and had been in labour three days before Moodelly saw her. She was found in a state of exhaustion, 'with the uterus prolapsed.' The labour-pains were very feeble and ineffective, and the membranes were entire. A grain of opium produced some sleep, soon after which the pains increased and she was delivered of a puny male child, which scarcely had power to cry and died in three days. The uterus was replaced and secured in position, and, when the report was made, the woman was doing well.

Dr. Webber and Surgeon-General Francis have reported¹ the case of a native, *æt.* 20, primipara, in whom *the spleen of the fœtus, being enormously enlarged, arrested delivery.* The dead child's abdomen was opened, when an immense hard tumour presented which had to be broken up before it could be removed.

Prep. 34, series 10, in Sibthorpe's Madras Catalogue. In a woman *æt.* 28, the head passed in thirteen and a half hours after the setting in of labour, but no further progress was made. The child's abdomen was found to be greatly enlarged. The child dying, delivery was effected by perforating the chest in the axilla and evacuating the contents of the chest and abdomen. The obstruction proved to be due to *kidneys enormously enlarged from cystic degeneration.*

Prep. 55 represents 'Cystic degeneration of kidneys in fœtus, requiring delivery by embryotomy.'

POST-PARTUM TETANUS. See the Chapter on Tetanus, page 417.

I have spoken, in the remarks on Tetanus and on Abortion, of the tendency which tetanus has to occur especially in cases where the emptying of the uterus has been caused by criminal violence; but this very serious complication is liable to occur after natural abortion and delivery both in Europeans and natives.

Having shown that tetanus is a very common cause of death in Bombay² Dr. Waring observes that, in the three years ending December 1853, no less than 232 women are recorded as having died from puerperal tetanus, mostly between the second and the tenth day—7 dying on the first day and 2, 9, 4, 1, 0, 0, 1, 1 dying between

¹ Cited from the 'Medical Press and Circular' in *I. M. G.* for September 1884.

² 'Notes on Some of the Diseases of India,' *I. A.*, No. 6, pp. 504 and 512.

the eleventh and eighteenth day, the liability greatly decreasing after the sixth day; the number dying during the first six days amounting to 145, leaving only 88 to be distributed over the remaining twelve days. Writing in 1856, Waring found that the liability to this complication appeared to be rapidly increasing. The larger proportion of deaths from puerperal tetanus takes place in the wet season, though a large number occurs in each quarter.

In his paper on tetanus as observed in the Calcutta Medical College Hospital, Mr. J. R. Wallace remarks that 'in women, idiopathic' [?] 'tetanus is very frequently associated with the puerperal period and with menstruation, for, in 52 females affected, 29 were either menstruating, or had just been confined at full time or had aborted.

In the following case, by Asst.-Surgeon Bhowanee Doss, we are left in doubt as to the cause of abortion; but it is clear that the *dace* was a suspiciously skilled manipulator. A young native woman aborted in the third month. The placenta being retained and becoming putrid, tetanus resulted. When the placenta was removed, a *pottee*—or small bag of cloth—containing saffron, bits of cloth &c., came away, having been placed there by the midwife to stimulate the expulsive power of the uterus. By evening, the trismus relaxed; and, by next day, tetanus and fever passed off.¹

At a discussion held by the Calcutta Medical and Physical Society in May 1835 Mr. Twining observed that numerous cases of tetanus, in native women, occur four or five days after delivery. He considered the cause to be excessive heat from charcoal fires in confined and crowded native huts.

Mr. A. H. Leith had two cases, at Poonah, in [European ?] women in which the attack occurred a fortnight after parturition. One of the patients recovered.²

Mr. Raleigh saw in Calcutta several cases of tetanus in native females after child-birth. They were generally fatal. He regarded the tetanus as 'arising from the inhuman customs they observe in their treatment after delivery.'³ Such is not always the case. Mr. W. A. Green⁴ narrates the case of a married [English] lady *æt.* between 30 and 40, the mother of several children, who had miscarried seven or eight days previously to the occurrence of tetanus. She had lately come off a journey, during which she had felt alarm and suffered from diarrhœa. On the night before the tetanus appeared, she had sat up in bed exposed to a cool current of air. There was no lochial discharge at the time of the attack. There were trismus, at first slight, going on to locked jaw; very severe opisthotonos, inability to swallow, and almost suffocative difficulty in breathing. She died on the eighth day of tetanus.

¹ *I. M. G.*, vol. viii. p. 321.

² *I. M. J.*, N.S., vol. vii. p. 709.

³ *I. M. J.*, N.S. (Finch's), vol. ii. p. 99.

⁴ *I. A.*, No. 11, p. 71.

Dr. Green narrates three cases in which native women were attacked with tetanus on the seventh, ninth, and ninth day after delivery. The tetanus proved fatal in two, four, and *cir.* fourteen days. One of these women was the subject of placenta prævia with severe and long-continued hæmorrhage. Dr. Green delivered by the feet. The labours of the other two women appear to have been unattended with difficulty.

Baboo Bindabun Chatterjee gives¹ the case of a Mohammedan labouring woman who was admitted to the Calcutta Medical College Hospital, on the twelfth day after delivery of her first child, in a state of tetanus. On the third day, the spasms had entirely ceased. She, however, sank from chronic dysentery on the sixth day after admission.

Baboo Shamloll Mullick reports² the case of a Hindoo woman, *æt.* 30, who recovered from puerperal tetanus under the use of chloral and bromide of potassium.

P. S. Mootoosamy observes that, in the Madras Presidency, 'Comparatively few females who are delivered in our public hospitals become the subjects of tetanus.' Not a single case of tetanus occurred in 5,794 women in the Reports of the Madras Lying-in Hospital. He attributes puerperal tetanus to the evil sanitary condition of lying-in females. The unfortunate woman, in an ill-ventilated, dark, and damp hut, 'is literally starved for the first three days after delivery.'³

Dr. Branfoot has lately remarked⁴ that probably septicæmia has often some action in causing tetanus. In several cases of so-called 'idiopathic' tetanus, he had found injuries to the cervix.

The facts which I have cited show that the tendency to this dangerous complication of labour ranges, at least, from Bombay and Poonah, in the West, to Dacca in the East, and from the North-Western Provinces southward to Calcutta and Madras.

PUERPERAL CONVULSIONS.—The presence of convulsions or eclampsia may be considered as the rule, rather than as the exception, in that miserable class of cases in which young and often immature native mothers, or those whose constitutions are broken by child-bearing, are first seen in the third or fourth day from the commencement of labour with the head tightly impacted in a pelvis of deficient capacity, or with preternatural presentations; uterine action having ceased for hours. In the 52 cases reported by Mr. Green, as representing most of the difficulties and dangers encountered in obstetric practice at Indian civil stations, and exhibiting 'the extreme instances,' I find that convulsions occurred in 15, and tetanus in 4. Undoubtedly, difficult labour is the most usual cause of puerperal convulsions; but, as is shown in some of Mr. Green's cases, convulsions may (1) precede labour, (2) occur early in labour, or (3) follow it.

¹ *I. A.*, No. 3, p. 471.

³ *I. M. G.* for November 1877.

² *I. M. G.* for 1877, p. 70.

⁴ *Ibid.* for November 1885.

The subjects of two of Mr. Green's convulsion cases were European ladies, who recovered. In a rather considerable experience among this class of patients, I never saw, or recollect to have heard of, any other cases. Two of Green's cases occurred in East Indians, who recovered. Of the 11 native patients thus attacked, 9 died.

P. S. Mootoosamy has observed¹ that puerperal eclampsia is of frequent occurrence in native females in Madras.

Recently, Surgeon Browning, of that city, has reported² a case in which *convulsions appeared thirty-two days after delivery*. The patient, a Hindu primipara, *æt.* 25, was demented for several days prior to the attack. For a fortnight after confinement, the urine was albuminous; but, in the attack, it was free from albumen. In each paroxysm, the left side of the body was more affected than the right. The paroxysms became more frequent and a *status convulsionis* was established, in which she died on the morning of the fifth day, of convulsion.

In Dr. Branfoot's 1,427 cases of delivery, in 1882, so frequently cited in this chapter, puerperal convulsions (eclampsia 3) occurred in 23. Of these cases, 7 were fatal.

In 1883, this physician had, in 1,896 deliveries, 22 cases of convulsions, 80 per cent. of which occurred in primiparæ. Eight cases were fatal.

Among the soldiers' wives there were, in 1880, 3 cases of puerperal convulsions, one of which was fatal. In the following year, there was not any case; in 1882, there were two cases, neither of which was fatal. In 1883, there was one which was fatal.

Dr. D. R. Thomson's Report of the Ramasawmy Lying-in Hospital, Madras, for 1882, gives 9 cases of eclampsia, with 4 deaths, in 477 deliveries. In this institution, in 1883, there occurred convulsions in 9 out of 442 deliveries. Of these nine, 5 proved fatal. Dr. Thomson observes: 'This is a very high percentage, and is only to be accounted for by the fact that all were out-cases, and chiefly among the better classes of Hindu women, who seem to be predisposed to it and are brought to hospital, after considerable delay and neglect, in a state of exhaustion; death often occurring a few hours after delivery.' Not less than 88·89 per cent. were primiparæ.

Post-Partum Hæmorrhage.

I have already mentioned that more or less anæmia is so general among women of all races and classes in India, that, throughout my active career, I never failed (1) to have the long sheet-binder spread across the bed, under the woman's body, immediately she lay down, so as to be able to use, with the aid of firm pads, any needful amount of compression at a moment's notice—always as the placenta was

¹ *I. M. G.*, vol. xii. p. 6.

² *Ibid.* for November 1885.

ready to pass; and (2) to have a full dose of ergot ready to be given immediately I found that the child was just about to be born.

In a well-reasoned paper on the use and abuse of ergot in labour, which he laid before the Calcutta Medical Society, in November 1880, Dr. Harvey observed that we value ergot merely as a preventive of hæmorrhage. What we want, after the child is born, is to induce strong, persistent, rigid contraction of the womb by which to diminish the calibre of its vessels and favour their occlusion. The special effect of ergot is just what we want now, and the more powerfully it acts, the greater is the security given against flooding; this use of ergot is, he thinks, hardly sufficiently recognised. The ergot is commonly not given until the flooding has begun, and here, though it often succeeds, it sometimes fails. As a preventive of hæmorrhage, it is best given just as the head is passing, and it has been the rule, for years, in the Medical College Hospital, to give a dose to all patients at this time. It begins to act at about the time of the separation of the placenta, and, on expressing the placenta, the hand following the uterus will find it firmly contracted, and in that condition it remains. He recently had a case where the patient, who had had severe floodings after each of three previous labours, and had been warned to have ergot, and iron, and ice, and sponges in the house, did not lose a wineglassful of blood altogether. Since he has adopted the plan of giving a dose of ergot in this manner, he has had far less post-partum hæmorrhage than he had before. In India, the relaxing effect of the climate makes this a much more frequent occurrence than it is in England, and it is therefore specially necessary to take precautions against it.

While I held civil medical charges, I never formed the purpose of remaining out of my station all night except once, and then, providentially, I altered my mind. I attended throughout that night a European lady, who must have died in the absence of proper assistance. She was a remarkably large and handsome person, between thirty and forty years of age, very pale, and evidently not strong. A European midwife, whom I had never seen before, was in attendance. I had separated the child and was placing it on the spot prepared for it, when the midwife shrieked, 'You have torn something away!' I was rather startled, more by the woman's piercing scream than from any idea of disaster. A large bag of amniotic membrane, filled with blood, had come down, and there was profuse hæmorrhage. Moderate stress upon the cord would not move the placenta. I therefore introduced my hand rapidly, and, finding that the placenta was partly detached and considerably adherent, I peeled it off. Hæmorrhage was now speedily arrested, but not before jactitation had commenced. She made a fair recovery, but dropped suddenly dead on board her husband's steamer, some months afterwards. We subsequently discovered that the midwife was a lunatic.

The returns show that, in 1880, 19 European soldiers' wives were

treated for uterine hæmorrhage (not sub-divided), with 1 death; the strength being 4,134. In the following year there were 24 cases, none fatal. In 1882, the number was 16, without any death: in 1883, there were 18 cases, one of which was fatal.

In 1,427 cases of labour, reported by Dr. Branfoot, as occurring at the Madras Government Lying-in Hospital, in 1882, there were 36 cases of post-partum hæmorrhage, of which one was fatal. These included all cases in which the loss of blood after the birth of the child was in any way excessive. In 29 cases, the loss of blood was not such as to retard the convalescence of the mother; in three cases, it was sufficient to affect the woman's pulse; and, in four, active measures were called for to control a considerable loss of blood.

The modes of delivery in these cases were:—by natural powers, 25; forceps, 9; version (forceps having failed), 1; version and craniotomy, 1. In 4 cases, solution of iron was used to arrest bleeding; in 3 others, hot-water irrigation acted efficaciously.

In like manner, Dr. Branfoot reported 38 post-partum hæmorrhage cases in 1883. In 27 of these, the loss of blood was trivial. In 5, it was sufficient to affect the mother's pulse; and in 6, active measures were called for to control a considerable loss of blood. The mortality was 1. In five cases, solution of perchloride of iron was used to arrest bleeding; and in two, hot-water irrigation was efficacious.

In 477 deliveries at the Ramasawmy Lying-in Hospital, Madras, in 1882, there were 6 cases of post-partum hæmorrhage, of which 3 were fatal. In 1883, there were 3 cases, none fatal.

I have already made allusion to the following very important case, which I saw with Sir Joseph Fayrer:¹ The patient was a young English lady, *æt.* 23, who had been married about ten months, and had resided in India four months. She was of a tall and sufficiently vigorous, though rather slight, frame; her general health was good, nor was there anything in her appearance suggestive of deficiency in vital force. She was said to have suffered severely from measles shortly before her marriage, and was considered to have been somewhat constitutionally weakened thereby. She had passed through the period of her pregnancy without much inconvenience, and had completed the full time when labour commenced. The progress of the first stage of labour was unusually slow, for, commencing on Saturday evening, it was not until Tuesday at noon that the foetal head passed through the cervix, and entered on the second stage of labour. But, as her constitutional powers were not depressed, no interference, beyond small doses of antimony to facilitate dilatation, opium to give rest, and chloroform occasionally, was considered necessary; and the result proved that this view was correct, for the second stage of labour

¹ It is given in full detail in Fayrer's *Clinical and Pathological Observations*, p. 584.

was completed within six hours, and she gave birth to a vigorous and healthy child without much difficulty and with little suffering, as she took chloroform. After the expulsion of the placenta, the womb contracted firmly, and, up to this period, there had been no loss of blood. Soon after the placenta had come away, the pad and binder were applied. Her pulse, which had been quick, sank to almost the normal standard. About thirty-five to forty minutes after the birth of the child, she said that she felt uneasy, and had a violent pain in her back. The pulse had suddenly quickened. The uterus was found to have relaxed and to be distended with blood. The clots were immediately removed with the right hand, the womb being grasped with the left. Ice and douches of iced water were employed externally, and iced water was injected into the uterus. Half a drachm of liquor ergotæ was given, and powdered ergot shortly afterwards. The magneto-electric current was applied, and the child was put to the breast. The relaxation of the womb which caused the loss of blood was sudden, but it was rapidly arrested; and though, in the first gush of hæmorrhage, a considerable amount, about two pounds, of blood was lost, there was no repetition of it. The uterus, after being relieved of the clots, contracted firmly, and remained so to the last. She was considerably depressed, but did not, at this time, lose the red colour of the lips and eyelids; the pulse was rapid and irregular, but her voice was good, and she seemed free from alarm when, in reply to her query, she was told that the bleeding had been controlled. She did not faint, neither did she manifest, at this time, the usual symptoms of dangerous hæmorrhage. She was quiet, and spoke calmly and cheerfully about herself. She was given brandy-and-water freely, and beef-tea and brandy; and mustard poultices were applied over the heart and solar plexus, and to the back. Brandy was also given in the form of enema, and hot bottles were applied to the extremities; but her condition did not improve. The pulse became weaker and more rapid and irregular; she was restless, and the surface of the body was bedewed with a cold sweat. The countenance began to change. These symptoms did not make their appearance for fully half an hour after the hæmorrhage had finally ceased. During the application of the magnetic battery, &c. she was in good spirits, held the wire with her own hand, and laughed at the nurse who held the other wire. Reaction never properly set in, she seemed to have no power of rallying, and, notwithstanding every effort, she gradually sank. The pulse occasionally rose slightly, giving a delusive hope of reaction, and for a few moments she slept; but, at last, the breathing became hurried, as though pulmonary obstruction was taking place from coagula forming in the right side of the heart. She became intensely restless, talked for a short time incoherently, and then sank and died quietly three hours and fifty minutes after the

birth of the child, and about three hours and a quarter after the occurrence of the hæmorrhage.

Sir Joseph Fayrer observes that 'the amount of blood lost was not so great as to give rise to dread of impending death. Much more has been lost in other cases, and yet perfect reaction and recovery have followed. But,' he adds, 'there are certain constitutions that seem to be endowed with but little power of rallying from a shock, even though slight, and in which the vital energy, though equal to all the ordinary emergencies of life, is inadequate to the task of recovery when any serious cause of depression has affected the nerve-centres. In such, no doubt, the great heat of a Calcutta May, and its terribly depressing influence must be an additional source of weakness, and a most important obstacle to recovery, when any such shock to the nervous system has taken place.'

I have always thought it a very noteworthy fact that this labour extended from the evening of the 22nd to that (time of death) of the 24th of May, a time which was, on that occasion, as it generally is, the very hottest and most painfully oppressive season in the Calcutta year.

It was, in fact, what I term 'heat-stroke weather.' In this case, there was not any symptom characteristic of insolation; but I fully agreed in the opinion that, had delivery occurred at almost any other season, the exhaustion would not have proved mortal.

Mr. A. R. Hall¹ has argued that post-partum hæmorrhage may be occasioned by tying the umbilical cord previous to the natural arrest of circulation between the mother and the fœtus. He therefore deprecates the too early application of the ligature to a still pulsating cord.

Considering how long the head frequently remains impacted in the narrow immature pelvis, and taking into account the violence frequently employed by native midwives, it is to be expected that sloughing of the maternal parts and, in exceptional cases of survival, *Vesico- and Recto-Vaginal Fistulæ* should result. Only too many reports like the following might be cited:—'Hindoo, has been in labour four days; contracted pelvis, no pains since yesterday morning; labia and vagina much swollen, torn, and bruised from manipulation by native midwives, slow delivery by craniotomy and laceration, death from exhaustion on the following day.' 'Hindoo, in labour three days; head of a dead child low in the pelvis; craniotomy. Four days after delivery, sloughing of vagina, which went on to produce recto-vaginal communication. Died 22 days after delivery.' 'Native woman in labour four days; head low down, tightly fixed at lower outlet of pelvis; fœtus putrid, all labour-pains had ceased for two days; the woman had been brought from a distance. Craniotomy. Sloughing between all the passages ensued, throwing them into one; the urine

¹ *I. M. G.*, vol. iii. p. 151.

constantly dribbled away. The woman had had fever and diarrhœa; ulcers formed on the nates from constantly lying in the wet. She was ill-looking after. Death in 16 days.' 'The woman had been four days in labour when seen. Head low down and impacted at the lower outlet of the pelvis. Craniotomy. Sloughing and inter-communication took place between vagina and rectum; urine also constantly dribbled away. Diarrhœa succeeded. Death on the 8th day after delivery.'

Prep. 31, Series 18, in the Madras Collection is 'Calculus extracted through a vesico-vaginal fistula, which was enlarged for the purpose. The nucleus is a plug of wood placed in the fistula by a native midwife about three months before. The flow of urine was checked by it for a few days, but the plug afterwards fell into the bladder.'

Early in 1884,¹ Dr. Harvey, of Calcutta, reported a case of vesico- and recto-vaginal fistula, in which he had resorted to the operation of closing the urethra and vulva for the purpose of causing the urine to be discharged *per rectum*. A Mussulmaanee, æt. 21, said that the midwife had used much force in delivering her, and that, ever since, the urine and fæces had escaped *per vaginam*. The communications were large. Dr. Harvey denuded the whole of the tissues round the orifice of the vagina for about an inch up the lower part of the vagina and up to the glans clitoridis. Three deep sutures were then passed, and a catheter was introduced *per anum*, through the hole in the rectum, into the bladder. The deep sutures were then tightened upon quills and two superficial ones put in. The case did well from the first. There was but little constitutional disturbance. The urine was allowed to dribble through the catheter for a week; a stopper was then introduced, and the water allowed to accumulate for an hour at a time. There was some diarrhœa on the third day, the stools passing without difficulty by the side of the catheter. The deep sutures were cut on the sixth day, to relieve tension, and were removed on the eighth, when the wound was found to have completely healed. The catheter slipped out of itself on the twelfth day, and was re-introduced. When the report was made, she could go fully two hours without a call to stool, and the urine and fæces passed together, but were under complete control.

Mr. J. Hinder reported² the case of a Mohammedan girl of delicate appearance, eleven years of age, with a recto-vaginal fistula, said to be the result of forcible sexual intercourse about two months previously. The opening was about an inch long, and caused considerable distress, by the escape of fæcal matter into the vagina. An attempt to close the aperture, by paring and metallic sutures, having failed, the sphincter ani was divided on Copeland's principle. The aperture contracted and gradually closed, the sphincter eventually recovering its

¹ *I. M. G.*

² *Ibid.*, vol. i. p. 156.

contractile power, and she was perfectly cured in a month and seven days.

Eversion of the Bladder is to be numbered among the cruel lesions inflicted by native midwives. Mr. McGann met with two cases.¹

The reporter cites Sir H. Thompson, who states, in 'Holmes's Surgery,' that only three cases had come to his knowledge, all in female children who, when first seen, were from two to four years of age; the condition having probably commenced at a very early age. The cases were so much alike that only one is fully detailed. A Hindoo prostitute, *æt.* 40, was admitted with famine diarrhœa. From the vulva, a pyriform tumour, about the size of a small orange, was seen protruding, with the mucous surface thickened and having a granular vascular appearance. The openings of the ureters were not distinguishable, and the urine 'dribbled, as it were, from the surface of the tumour.' The uterus was prolapsed, the os being visible at the vulva, and it had a fold of vaginal mucous membrane both before and behind it. She said that, about seven months previously, she became pregnant, and took medicines, with a view to procure abortion, which caused expulsive pains so strong as to produce the prolapse of the uterus and eversion of the bladder. The bladder was reduced under chloroform. 'The urethra was found to be much lacerated and dilated; with a diameter of over three quarters of an inch. Three days later, eversion again occurred; the bladder was easily reduced. Up to the time of her death from the diarrhœa, three weeks afterwards, there was no return of the eversion. In the other case, the bladder was reduced and 'while the distended and lacerated urethra was contracting' the woman absconded. She was very unintelligent, but stated that the eversion came on after parturition.

Prep. 15, Series 11, in the Madras Museum, is 'mucous membrane of the bladder, which protruded through the urethra, and was removed by forceps.' The woman had been confined a few days previously at the Lying-in Hospital. There were not apparently any symptoms indicating bladder-mischief. She made a good recovery.

Retained Placenta.

In the case of an English lady whom I attended at Howrah, the placenta was normal and whole, and there was not any post-partum hæmorrhage. About three weeks after delivery, she passed a supernumerary placenta, *placenta succenturiata*. Its size and form were nearly those of the pancreas. It was absolutely free from decomposition.

Ten ordinary cases of *retained placenta* occurred in the 1,427 deliveries at the Madras Lying-in Hospital in 1882. In eight, there

¹ *I. M. G.* for 1879, p. 46.

was simple retention, and in two there was morbid adhesion. Seven were delivered by the natural powers, and one by forceps. Mortality, one. This woman was brought in with retained placenta, having been confined four days previously at her house. Fever was present on admission, and the placenta was putrid. She died five days after admission. This case was one which could hardly occur outside the province of Indian *daces*. The patient was a Mohammedan, *æt.* 18, in her first pregnancy; admitted after having been confined, at full term, five days previously. Attempts had been made, by native midwives, at her home to remove the placenta, during which the cord broke. The temperature was 100°, pulse 124, skin dry, tongue furred and dry, abdomen tympanitic and tender, discharge from uterus dark and extremely offensive, uterus large, reaching up to umbilicus, vagina very hot. Under chloroform, the placenta was removed entire, some blood being lost during the emptying of the uterus. The uterus was irrigated with phenyle solution (1 to 100), and she was ordered quinine and chlorate of potash. The fever continued with offensive discharge and tenderness over the uterus. The cervix, on *p.-m.* examination, was found to be in a sloughy condition.

In 1883, nine cases of retained placenta were treated here. In seven, there was simple retention, and in two there was morbid adhesion: six of the women were confined in their homes and were brought in with the after-birth retained. The remaining three were delivered in hospital by natural powers. Mortality of mothers, *nil*.

Puerperal Fever.

I have spoken of Puerperal Fever and Malarial Post-Partum Fever under the head of Malarial Cachexia.¹ The returns show that, in 1880, there was only one (fatal) case of puerperal peritonitis among the 4,134 European soldiers' wives in India. There were three cases of puerperal fever, one of which was fatal. In the following year there was again only one case of puerperal peritonitis, which recovered. There were five cases of puerperal fever, all fatal. In 1882, puerperal peritonitis is not mentioned. There were seven cases of puerperal fever, six of which were fatal. In 1883, there were two cases of puerperal peritonitis, of which one was fatal; and four cases of puerperal fever with three deaths.

Dr. Branfoot states that 'All cases in which febrile symptoms continued for more than twenty-four hours, and which had an offensive lochial discharge, as well as those cases of continued fever after delivery, in which the cause was doubtful and might be attributed to blood-poisoning, are included,' in his Madras Lying-in Hospital report for 1882, under the head of 'Puerperal Fever. With much deference,

¹ Pages 271-72.

I do not quite admit the correctness of this arrangement, seeing that it tends to place different states of disease under one heading, and that, while true puerperal fever has a distinct tendency to kill, there occurs in many Indian cases, a post-partum fever, of malarial origin, which, although attended with very pronounced symptoms, is a by no means dangerous complication. Dr. Branfoot says, 'thirty-six of the fifty were admitted with fever before delivery.' This is not exactly 'Puerperal Fever.' The able reporter does not allude to Erysipelas, but we find this significant remark in the report of the Madras General Hospital for the same year: 'Erysipelas is rare in Madras, and never attacks patients in hospital.' There appear to have been fifty of these fever cases during the year in Dr. Branfoot's wards: of these ten were fatal. Dr. Branfoot well illustrates the tendency of puerperal fever to depend upon septicæmia from the retention of decomposing coagula, &c.; its proneness to occur and spread in certain hospital localities; and the practicability of checking it by isolating the patients in separate buildings.

In the 477 deliveries at the Ramasawmy Hospital that year, there were three deaths from puerperal fever and three from puerperal septicæmia.

During 1883, there occurred, in Dr. Branfoot's wards, three fatal cases of puerperal peritonitis, and eight of puerperal septicæmia. At the Ramasawmy Hospital there were, in 1883, from puerperal fever, three, and puerperal peritonitis, two deaths.

In a paper on puerperal fever among Parsees in Bombay,¹ Mr. Teemoljee Nariman attributed the occurrence of this condition to the national practice of confining the mother in a room on the ground floor for forty days after delivery. In discussing this question, Mr. Henry Cook said that the women of the poor were thus 'confined, at a most critical period of their existence, in horrible dungeons surrounded by bad drains, where living at any time is misery.'

For the *Association of Puerperal Fever with Erysipelas* in India, see p. 321.

The parturient woman's condition is, of course, most dangerously complicated in the presence of either *Cholera*, *Remittent Fever*, or *Dysentery*. I have spoken of the two latter conditions under the heads of Malarial Post-Partum fever, p. 272, and Dysentery, p. 154. I never knew the collapse of cholera continue so long as in the case of a young Hindoo lady who was attacked when nearly at her full time. There was no radial pulse for nearly three days before she sank (this singular type of cholera was then prevalent in Calcutta), she died undelivered. In describing cholera, I have alluded to the case of a poor European woman who died of cholera in my ward not long before I left India.

¹ *I. M. G.* for September 1884, p. 270.

She was in the second month of pregnancy. A tendency to abort caused me anxiety, but this accident did not occur. In dysentery, the tendency to abort is very strong—doubtless in consequence of rectal irritation and low vitality.

Prolapsus Uteri.

I have already spoken (p. 761) of cases in which labour is complicated with procidence of the uterus. In India, prolapse of this organ is very frequent among young women, of all races and classes, who imprudently begin to go about in eight or ten days after their first or second confinement.¹ This condition may generally be cured by steadily maintaining the recumbent position—latterly on a couch—for at least a month after each subsequent delivery.

In the four years 1880–83, the numbers of European soldiers' wives treated for Prolapsus Uteri were 14, 12, 15, and 12. In all probability, the returns do not by any means adequately represent the suffering which exists from this cause.

The cases of Prolapse treated in the Gynæcological Wards of the Madras Lying-in Hospital in 1882, were, in 109 Europeans and East Indians, 6; and, in 167 natives, 6.

PLACENTA PRÆVIA of course occurs in both Europeans and natives. I had some very anxious cases. In one of latero-cervical placenta a wretched Irish barrack-woman, who professed to be a nurse, nearly caused the death of my patient by mismanagement of the binder after delivery. Happily, the lady still lives and her son is a medical officer in India.

The returns show that, in each of the years 1880 and 1881, a case of placenta prævia, in which the mother recovered, occurred among the soldiers' wives in India. There was not any case in 1882 or in 1883.

Four cases of *Unavoidable Hæmorrhage* occurred in 1882, in Dr. D. R. Thompson's Ramasawmy Hospital in 477 deliveries. All of the mothers—none of the children—survived. In 1,427 deliveries at the Madras Lying-in Hospital during the same year, there were twelve cases of unavoidable hæmorrhage, in one of which the mother died. Two of these cases were delivered by forceps; two by podalic version; one by podalic version and forceps; one by podalic version and craniotomy; one by cephalic version and craniotomy; two by extraction and forceps; one by cephalotripsy; and two by the natural powers. Of the children, five were born alive, four were still and three were

¹ It would almost seem that Englishwomen inherit this error from their ancestresses. Von Raumer gives the following, in a description of England in 1551 by Petruccio Ubaldini: 'In childbed the women keep state with very white linen; some rise very soon, but go to church at the end only of twenty days, to return thanks.'

putrid. Only one case of *Accidental Hæmorrhage* occurred that year at the Ramasawmy Hospital, fatal to the child. At the Lying-in Hospital, eight cases of accidental hæmorrhage were reported, seven were delivered by natural powers and one by forceps. Two children were born alive, four were born still, and two were decomposed. One mother died.

In 1883, there occurred in Dr. Branfoot's hospital eighteen cases of *unavoidable hæmorrhage*, one of which proved fatal. Two fatal cases of *accidental hæmorrhage* (one twins) and one fatal case of *placenta prævia*. At the Ramasawmy Hospital, there were one case, fatal, of *accidental*, and two of *unavoidable hæmorrhage*.

PHLEGMASIA DOLENS.—Only two cases occurred in India within my own observation—both in European ladies. In both recovery took place; but, in one there occurred, about three weeks after delivery, an attack of cardiac difficulty, fought through in a quarter of an hour, which was, I feel certain, due to the detachment of an embolus. Dr. Vincent Richards reported¹ the case of a native woman who died suddenly of cardiac embolism a few hours after delivery. Among the European women, two cases of phlegmasia dolens occurred yearly in the three years 1880–82—none in 1883. One of these proved fatal. I do not find any notice of this lesion as occurring in the 2,061 cases of delivery and diseases of pregnancy treated in the two Madras Lying-in Hospitals in 1882.

Since my return to England, I have seen a very remarkable case in which, according to my judgment, the sequence of morbid actions has been as follows. A lady between 50 and 60 years of age was delivered of her only child in India some thirty-six years ago. Since then, she has suffered from various pelvic difficulties, of which it is needless to say more than that, for many years, she displayed great power of recovering from them. More than three years ago, violent pain, at or about the sacral promontory, caused so much steady distress that she took to her bed and has not left it since. Shortly after this, it became evident that the flow of blood through the inferior cava was interrupted low down, and it was clear that this duty had, at least in a very great measure, been taken by the external epigastric veins, the vastly enlarged canals of which convey an upward current, *vide* p. 502. With this change, great hypertrophy of the integument of the abdomen and mammæ has become established: the pain in the back has been less complained of; but, doubtless in consequence of the abnormal state of the venous circulation, a tendency to syncope, precordial uneasiness, and distress after taking food have occasioned almost constant debility and suffering. Various ideas suggested themselves to my mind—malignant plugging of the cava, uterine fibroid pressing backward on this vein, &c.—but there was no evidence

¹ *I. M. G.*, May 1872, p. 106.

of the existence of any uterine disease or of tumour there or elsewhere, and there was not the slightest appearance of malignant cachexia. She had been ill nearly two years when I saw her, although I had often heard of and reflected upon her case. Understanding that there was some oedema, I said, immediately the feet were uncovered, 'Did phlegmasia dolens follow her confinement?' The reply was in the affirmative. The left leg is still large, the right has been liable to swell ever since the birth of her child. I feel no doubt whatever that her present condition has been occasioned by increased obstruction, probably by thrombosis, occurring in the faulty cava.¹

Pelvic Peritonitis, Cellulitis, and Hæmatoma associated with Pregnancy.

Cases of what we used to speak as 'Pelvic Cellulitis' used to occur within my own Indian experience in women of various races. The returns show only two cases of this lesion, one fatal, as occurring among the wives of European soldiers in the four years 1880-83. In the gynæcological wards of the Madras Lying-in Hospital in 1882, there were six cases of pelvic cellulitis among 109 European and East Indian patients, and 14 cases in 167 natives. Baboo Ram Chunder Mitter reported² a case of retention of urine which he attributed to pelvic cellulitis. It is noteworthy as an illustration of the practice of native midwives. A Mohammedan woman, *æt.* 20, not having menstruated for three months, and considering herself pregnant, consulted a daee who, in order to re-establish the menses, applied some soft, paste-like stuff apparently around the os uteri. 'The swollen and oedematous condition of the parts, consequent on the inflammation of the cellular tissue of the pelvis, together with the paste kept inside, excited so much pressure on the urethra as to cause retention of urine, which was of three days' standing when the sub-assistant-surgeon saw her. More than six pints of urine were drawn off. The paste was removed, and the vagina, which was hot, red, and swollen, was syringed. There was considerable constitutional disturbance, and the use of the catheter had to be continued for three days.

For two remarkable cases of Pelvic Abscess, supervening on Hæmatoma, in native women who had not been pregnant for years, see 'Diseases of Women' p. 728. M. Gallard, in his essay 'Du Phlegmon Periuterine,' believes pelvic cellulitis to be very rare, except in puerperal

¹ For 'Thrombosis of the veins of the lower extremity, resembling phlegmasia dolens, as occurring in the male subject in India,' see p. 254 and *C. M. T.*, vol. vi. p. 495, by Mr. Boswell; *ibid.* vol. viii. part 2; Appendix p. 205, by Mr. Taylor and *Madras Quarterly Medical Journal*, vol. iv. p. 347.

² *I. M. G.*, vol. ii. p. 273.

cases. This does not coincide with the experience of Dr. David Boyes Smith,¹ who, while writing, had two cases of the kind in the Calcutta Medical College Hospital, and had recollection of a vast number of others. He was, however, prepared to allow that suppuration is more common and more fatal in puerperal than in non-puerperal cases. In India, where we do not, as a rule, see such non-puerperal cases in their early stages, Dr. Smith has observed that it is very common indeed to find that they have run on to the formation of abscess. He had no recollection of any single case of this kind, within his own experience which proved fatal, although he remembered a good many sad and fatal puerperal cases, the result of tedious or instrumental delivery. McClintock mentions that, out of 70 cases of puerperal pelvic cellulitis, only two proved fatal. Dr. Smith's experience was far more unfavourable than this. This might be expected in India, and especially among native patients.

Dr. Smith was surprised that more had not been written on this subject in India, seeing how commonly the lesion engages our attention by the bedside. Within that year, in the M. C. Hospital and out of it, he must have seen at least fifty cases, either simple or complicated by the subsequent formation of abscess. The little firm almond-shaped swelling in the vicinity of one of the broad ligaments, most generally on the left side, or the firm little tumour felt through one or other of the vaginal walls, at once attracts attention to a condition of parts which was formerly overlooked.

Where there is any reasonable hope for the resolution of an abscess or a hæmatoma, Dr. Smith would most certainly never think of using a knife or a trocar. But, in cases where resolution is unlikely to occur, where the constitutional symptoms are urgent, and where there is any indication of pointing at any part of the tumour, particularly if this occurs between the rectum and vagina, he would never hesitate to do what he has done in a good many cases, to make an opening where it seems most advisable to do so. He could by no means agree with Becquerel when he says 'the abscess is never to be opened, even though its apparent pointing in one situation should seem to invite interference.' Matthews Duncan, Professor Braun, and Professor Simpson have all advocated the practice of making the necessary openings in suitable cases. The latter authority cites a most instructive case in point. He saw a patient with Dr. Ziegler, in whom pelvic abscess threatened to burst spontaneously; they thought that it would find an exit for itself into either the rectum or the vagina. Within twenty-four hours, the abscess did burst, but it was into the peritoneum: the patient died in consequence. A timely opening would, he adds, have in all probability saved her life.

¹ 'Commentary upon a case of combined Pelvic Hæmatoma and Pelvic Abscess *I. M. G.*, vol. i. p. 150.

Disorders of Lactation.

In old times, the prophylactic measures chiefly relied on in cases where *Mammary Abscess* (which is rather frequent in India) appeared to be threatened, were the use of tartarised antimony and of prompt venæsection, 'with a full stream from a large orifice,' say to 'sixteen ounces.'¹ Doubtless such treatment afforded great present relief by lessening distension and pain; but Mr. L. W. Stewart justly remarks,² 'I am aware of many ladies who, from the effect of confinement to the house, low diet, and depletion for mammary abscess, were obliged to return to England.' Mr. Stewart advocates strapping. Attention to the state of the nipple and careful use of the exhausting glass will generally prevent the occurrence of abscess.

All over India and Burmah, native women suckle their children until they are five or six years old, chiefly with a view to averting pregnancy, a plan which often fails. Dr. Waring saw in Burmah, a six years' old 'infant,' who varied the amusement of sucking by an occasional whiff of a cigar. The gipsy women of India, who are engaged as dancing-girls and tumblers, permit their children to remain at the breast until they are five or six years old. Mr. Mitford states that the same practice obtains among the poor in Japan, it being by no means uncommon there to see a woman suckling a hulking child from four to seven years old. A woman born and brought up in India, but apparently of pure European blood, was admitted to my ward with unmistakable evidences of starvation. This was due to her obstinate pertinacity in suckling a strong active girl of five. She ultimately sank. When famine cases were admitted to the Calcutta Medical College Hospital, my friend Dr. Francis, while acting for me, had a most interesting photograph of a starved Bengali mother and her infant, taken a few hours previous to the woman's death by inanition. The emaciation of the woman's frame is extreme, save that the breasts are very large, and are evidently full of milk. All that the poor creature has of nourishment and life evidently goes to her offspring, which appears to be in fair condition.

An assistant-surgeon³ relates the case of an emaciated and anæmic native woman, *æt.* 20, who had been suckling her child with both breasts up to the time of admission. For six months, a large swelling had been forming in the left breast. Milk oozed from the nipple on pressure. The surface was vascular, soft, elastic, and glistening. There was fluctuation. Puncture gave exit to 'nearly thirty-six ounces of pure thickened milk.' For six or more days, about an ounce of milk was daily pressed out of the opening. Under the use of a zinc injec-

¹ Dr. D. Birch, *I. M. J.*, N.S. vol. v. p. 607.

² *I. A.*, No. 9, p. 273.

³ *I. M. G.*, vol. iv. p. 55.

tion the cyst contracted, and she was discharged much improved in health in a month.

In his account of native practice in Rajpootana, Surgeon-General Moore notices 'a case of destruction of the female breast, and almost loss of life following milk abscess.'

Lactation in New-born Infants. A tumid state of the breasts, sometimes a few days after birth, has been noticed by several observers, especially by Ramsbotham¹ who described the breasts as being tumid, hard, and painful, and turgid with a serous fluid that may be squeezed through the nipple. He evidently regarded the nurses' idea that this secretion is milk, which requires to be evacuated by friction and pressure, as a vulgar and mischievous error. He describes both organs as sometimes acquiring a very considerable size, so that the form resembles that of a woman with a very full neck. He considered that the affection is inflammatory, but held it to be of no moment, if nature be unmolested, the swelling subsiding in a week or a fortnight. He had seen this oftener in boys than in girls.

Dr. O'Connell Raye² was asked by a sergeant's wife to see her baby, as she had been obliged to draw off the milk from its breasts daily since its birth. The infant—seven weeks old—was a healthy, well-formed girl. Both mammæ were large, measuring nearly two inches in diameter, and having small partially-developed nipples, from each of which the mother, in his presence, pressed out somewhat less than a teaspoonful of milk in a full jet. She said that she used to remove a similar quantity daily by squeezing the breasts between the finger and thumb, and that, if not so relieved, the breasts became 'hard as a stone' and very tender. The milk was examined by Dr. D. Cunningham, who found that it had the appearance of healthy milk, except that the molecular oil was in larger quantity than usual. The father was an Eurasian; the mother was born in India of European parents.

The family of my second midwifery case in India (an English officer's wife, probably not of unmixed race) were much exercised by fear that *Puerperal Mania*—from a severe attack of which she had suffered after the last confinement—would recur. It did not. I heard of but few cases in India. The returns show that, in the four years 1880–83, there occurred six admissions for puerperal mania; one in 1880; one in 1882; and four in 1883, among the wives of European soldiers.

Note on Midwifery Practice among the Natives of India.

Although my own personal concern in Indian medical affairs ceased many years ago, the well-intentioned movement which has been for some time in progress for sending qualified female obstetricians to the East, has caused me a great deal of anxious thought.

¹ *Medical Gazette*, September 1835.

² *I. M. G.*, vol. v. p. 171.

In England, within the last fifty years, the midwife has been changed from a *quasi*-professional person into a subordinate nurse. Unquestionably, the ignorant and absolutely unteachable *dace* still holds unchecked sway in millions of native households. The intention of sending female aid from home, with a view to the abatement of this evil, is undoubtedly prompted by benevolence; but I perceive clearly that, unless it be conducted with great tact and care, by highly-qualified and judicious ladies, the measure will prove retrogressive, and will result in more harm than good. Would the excellent ladies who are earnest in promoting this mission and the majority of their countrywomen advocate the introduction of this system at home? Those who have originated this experiment possibly would, but no one else would entertain the idea for a moment. Otherwise, the project of educating medical women for home work would advance far more rapidly than it is doing at present. In every one of the many years of my Indian experience the aid of medical men in the treatment of native women was more and more sought for by the natives; and I believe that the number of those natives who would rather allow death to occur than call for the aid of a medical man, is yearly becoming more and more minute. Is it wise to check this advance in humane and saving enlightenment by sending out medical women, however highly qualified they may be? Let us take a single illustration of work done in the Presidency of Madras. In 1852, 504 women were confined in the Madras Lying-in Hospital.¹ In 1883, 2,338 in-patients and 21,426 out-patients, chiefly native, were treated at that and the other admirably conducted hospitals for women in the city of Madras. In 1883, under a system which was admitted to be susceptible of great improvement, we are told that the number of labour cases treated throughout the Madras Presidency, under the supervision of our district surgeons, had 'risen from 4,740 in 1882, to 5,544 in 1883.' It is added 'the number is less than it was in 1881, but is nearly twice as great as it was five years ago.' Does true humanity demand that such progress should be discountenanced and checked in its present vigorous and most hopeful growth by this latest offspring of mistaken philanthropy?

DISEASES OF BONES AND JOINTS.

In India there is undoubtedly great exemption from several principal forms of disease of the osseous system prevalent in Europe. Rickets, general mollities ossium, tuberculosis of bone, very rarely afflict the natives of Hindustan. Malignant disease is not rare, especially in the young. The recklessness with which syphilis is treated with mercury in native practice leads to a large amount of bone-

¹ Dr. James Shaw's Report, *I. A.*, vol. i. p. 144.

disease. Ordinary malignant and other diseases of the maxillæ frequently occur. Little needs be said here regarding those lesions with which all surgeons at home are familiar.

I have spoken, under the head of 'Hospitalism,' p. 330, of *Osteomyelitis*, as it long prevailed in the Calcutta Medical College Hospital.

There is an article on *Abscess* in the head of the tibia in Fayrer's 'Clinical Surgery in India.' Prep. 61 in Ewart's Catalogue is a 'longitudinal section displaying the oval-shaped cavity of an abscess in the cancellated structure of the upper third of the shaft of the tibia. About three inches of bone are necrosed.'

No. 34 is a very remarkable and beautiful preparation contributed by Dr. Theodore Duka. A very large accumulation of new bone occupies the upper part of the middle third of the right tibia, the consequence of abscess in that bone. It measures seventeen inches in transverse, and twenty-two inches in vertical circumference. At the first glance, it has much the look of a diseased skull. It is grooved externally for the reception of the fibula, which has been considerably bent by the pressure of the growing mass. Here the new bone is comparatively smooth and thin. In front, and on the inner aspect, it is more irregular, nodulated and perforated. One opening is large enough to admit a child's fist. Looking into the cavity from this point, numerous truncated developments are seen. Longitudinal section brings these still more clearly to view. The subject was a native. Amputation was successfully performed below the knee. This specimen interested me greatly, as displaying the extreme development of chronic abscess in a bone-shaft where surgical assistance was long unsought.

Prep. 35, from a native, displays 'a large development of new bone at the lower third of the left tibia, the consequence of interior abscess. It is much nodulated, and perforated with numerous holes, involving complete destruction of the ankle-joint and osseous union with the lower four inches of the fibula. Its cavity is very irregular and occupied by bony processes. It measures fifteen inches in transverse, and twenty in vertical circumference.

Cases of extensive *Necrosis* are by no means rare in ill-fed natives, especially as results of syphilis and mercury, as when salivation causes necrosis of the alveoli, and, where the unfortunate survives, atresia oris. *Necrosis* is a frequent sequel of *Smallpox*. This condition is illustrated by Prep. 1210. 'Three sequestra, each about four inches long, from the radius of a young native lad, consequent upon inflammation in the debilitated state of constitution following smallpox. Necrosis of the bones of the face, &c. is frequent in cases of cancerum oris (malarial phagedæna) as in the case of my little patient (*vide* p. 258), in whom what would have been, in a dog, an intermaxillary bone, with all its processes, came away entire.

Prep. 1,265 always appeared to me to represent the case of the

man whose *two* left tibiæ John Hunter used to exhibit. It was presented by Dr. Rose, of Penang. It is described as 'Necrosis of the greater portion of the shaft of the right tibia, from the upper to the lower epiphysis. The dead bone is enclosed posteriorly and laterally by new bone, which is continuous with the head and inferior extremity of the tibia, but it is open in front. It is quite loose, but it is so incarcerated that it cannot be dislodged through any of the openings. A good deal of new osseous structure is thrown out around the fibula, which, at the lower three or four inches, is ankylosed to the tibia.' It appears probable that this unfortunate native did not seek surgical aid. If the necrosed shaft had been removed in time, the new bone which surrounds it would doubtless have become moulded into a strong and very efficient tibia.

Mr. McReddie¹ had under treatment a native lad, *æt. cir.* 14, in whom, after a sprain, the shaft of the tibia perished by *Acute Necrosis*. Upon the teaching of Professor Lister in the 'British Medical Journal' for Aug. 1, 1868, he determined to employ carbolic dressing, and to await the absorption of the sequestrum. In less than three months, the sinuses all fairly closed, and the skin completely cicatrised. The limb had become much smaller, walking was painless, and he was stout.

Dr. George Mackay has reported² two very interesting cases: (1) that of a native lad of 15, in whom there was a sequestrum, which appears to have consisted of the 'whole of the shaft of the tibia *with its superior articular end.*' 'A new tibia had been nearly formed from the head downwards to within — inches of the lower extremity.' (2) In a native boy of 11, who had fallen out of a tree, there was a protruding sequestrum about two inches long, a portion of the humerus divided off between its surgical neck and the point of insertion of the deltoid muscle. There had been a compound double transverse fracture of the bone, the upper and lower ends of the bone had united, allowing the isolated portion of the shaft to be thrown off.

The *vis medicatrix Naturæ* is very strongly displayed in neglected cases of compound fracture and of spontaneous amputation brought to our dispensaries from remote districts. In the mango season, we always expect to see injuries to the wrist and arm in Bengali boys who have fallen out of mango-trees.³ At Chittagong a boy of twelve was brought to me with the ends of the radius and ulna, with about two inches of their shafts, protruding at the inner aspect of the wrist. As the boy did not appear to suffer, and as separation of the black necrosed bones was evidently going on, I did not interfere with nature's reparative work. I was soon able to draw out the protruded bones; healing was rapid, but a very inefficient wrist remained.

¹ *I. M. G.*, vol. iv. p. 141.

² *I. A.*, No. 4, p. 287.

³ Just as an old chronicle tells us that the little Cockneys used to climb and fall when robbing the apple-orchards of Paternoster Row.

I saw nothing and heard little of *Mollities Ossium* in natives. A case, occurring in a native girl, will be found reported in the 'I. M. G.' for 1882, p. 253.

Prep. 1,207 displays the bones of the forearms of a native subject from the dissecting-room, twisted, shortened, and atrophied, with a certain degree of softening. 'The lower ends of the ulnæ have lost their usual appearance. The bones of the lower extremities were similarly affected.' We often see, in the professional beggars of India, atrophy and other deformities of limbs, which have every appearance of having been artificially produced in early youth by binding, &c. These deformities are often most extraordinary, as in a young girl whose mode of progression was by hopping on a vigorous limb for a few yards, and then going down and scrambling rapidly upon her hands and a lower extremity about two feet long. Of course such cases are of very little surgical importance. The following illustrates this class of deformities: Prep. 1,293, Carpal and metacarpal bones of of a Hindoo Faqueer. There is ankylosis of the metacarpal bones of the fore and middle fingers to the trapezoid and os magnum, and of the first and second phalanges of the same fingers to each other.' I recollect one of these men whose atrophied and ankylosed arm stood, evidently by no muscular effort, straight up above his head like a withered branch.

Ewart's Catalogue describes several specimens of exostosis—especially 238, 'Exostosis of the lower jaw of a native, *æt.* 27 years. The disease was of 10 years' standing. The whole of the growth, with a portion of the jaw, was successfully removed.' 239. 'A bony tumour on the left side of the dorsal vertebræ, which pressed upon and obstructed the descending aorta.' I have cited Dr. Duka's case of pedunculated ivory exostosis of the antrum, broken off and lying loose in the cavity, at page 533.

Hypertrophy of Bone is represented in Prep. 6. 'Enormous hypertrophy and increased density of a portion of the calvarium. Behind the frontal eminences the section is very dense and measures almost three quarters of an inch in thickness.' 7 'Remarkable hypertrophy of the bones of the skull of a child who died of hydrocephalus.'

Atrophy of Bone.—Prep. 19 represents 'Extreme atrophy of the calvarium. Both tables and the intervening osseous tissue are remarkably attenuated, and the external and internal surfaces are smooth and shining. The parietal and frontal bones are not more than the tenth or eighth of an inch in thickness.'

24. 'Extreme mollities or *Fatty Atrophy* of the right fibula. The whole of the earthy matter of the shaft has disappeared, and, at the extremities of the bone, some has been absorbed and replaced by fat. The shaft is now represented by a mere string of fibrous tissue. There

is caries of the outer head of the tibia, implicating the marginal portions of the knee-joint.

Preparations 128 to 131 are good illustrations of *Fatty Degeneration of Bone*, see 'Ainhum,' page 380. Before considering these specimens further, I shall cite a valuable observation by Mr. McReddie¹ who describes a disease not unfrequent in the North-Western Provinces and, he believes, in the Punjab. It is confined to the wrist and ankle, and does not seem to invade the larger joints. It consists in softening of the bones, due to partial displacement of the earthy matter by a fatty substance, or in fatty degeneration of bone. The form and natural size of a bone are preserved, but all its blood-vessels are occluded and destroyed, and it can be cut with facility with an ordinary scalpel—the knife encountering a gritty sensation. The section appears yellowish and greasy-looking; its structure is compact and firm. In firmness and appearance, it is very like fresh cheese. Cartilage partakes of the same degeneration as bone. The joint looks swollen from superficial deposition of fat; and there are sinuses which discharge small quantities of purulent matter of very low type. The disease is generally painless, the nerves partaking of the same disorganisation as the blood-vessels, until sloughing of the superficial tissues occurs. The reporter has not seen cases in persons under thirty years of age. The disease has not an inflammatory or strumous origin. Its subjects have all along enjoyed good health. A probe introduced into the sinuses will not encounter carious bone, for the disease is not caries, but there will be resistance as if from coming against cartilage. In fact, all the structures entering into the formation of the joint, bone, cartilage, synovial membrane, blood-vessels, nerves seem to undergo fatty degeneration. There is never any attempt at cure: amputation affords the only hope of recovery; but, unfortunately, few submit to this. Mr. McReddie gives the case of a Hindoo, *æt.* 40, in whom this disease had commenced six months previously without any assignable cause. On examination of the amputated limb, it was found that the bones implicated were the ends of the tibia and fibula, all the tarsal bones, and all the metacarpal bones to within a short distance of the toes. All the structures were quite exsanguine, and the tarsal bones were as if fused into one mass. The foot was enlarged by superficial deposition of fat: there was no caries or necrosis of any of the bones. The man made a good recovery and had an excellent stump.

The preparations already cited appear to afford illustrations of this condition. Prep. 128: 'Longitudinal sections of two metatarsal bones removed from a native male about twenty-five years of age, whose corresponding toes had dropped off from dry gangrene; and of another metatarsal and the attached phalangeal bones of the same feet,

¹ *I. M. G.*, vol. i. p. 62.

illustrating extensive fatty degeneration induced by imperfect nutrition consequent on arteritis and complete blocking up of the principal vessels by fibrine. The cancellated structure is almost wholly occupied by fat. The cortical layer of the bone is much attenuated. So soft were these bones that the sections now presented to view were as easily made with a scalpel as if they had been made through a piece of cheese. On microscopical examination, a great quantity of fat-globules was found. The soft parts of the Haversian systems were almost completely replaced by this fat. The foot was removed by Dr. Fayrer in 1864. No ligatures were required, and no sutures were employed.'

'130. Part of the first phalanx of the great toe of a woman, *æt.* 50, with fragments sliced away from the head of the first metatarsal bone, displaying an immense deposition of yellow-coloured fat occupying the soft parts of the cancellated structure. The cortical portion of the bones has been almost completely destroyed by fatty degeneration. The sections now presented were made by the scalpel as easily as if the bone had been replaced by cartilage. The patient had undergone an operation in the Gow Kana Hospital eight or nine months previously. The wound healed, but the cicatrix had been subject to ulceration and pain ever since. Dr. Fayrer removed the diseased parts, 1864, leaving sufficient integument to cover the bone thoroughly.'

'131. Leg amputated below the knee on account of the supposed removal of the largest part of the foot by an alligator. There is a cauliflower-looking excrescence, as large as an orange, over the situation of the astragalus. A section of the tibia, astragalus, and os calcis has been made, showing advanced fatty degeneration in each.'

Dr. R. Macleod, has described and figured¹ a remarkably curious case of absorption of the phalangeal bones of the hands of a Sonthali man, *æt.* about 23. The toes were affected in less degree. Only the thumb and ring finger of the left hand appear to have escaped. The description of a thumb and finger will suffice. The metacarpal bone of the former is absorbed, and the first phalanx dislocated on the palm and very mobile, but there is no pain. The phalangeal articulation is partially ankylosed, and the head of the first phalanx thickened. The first and second phalanx of the index finger are absorbed, and the third is partially so. The skin is healthy, and is thrown into folds. There were, at first, pain and swelling, passing off in a month, of the affected parts, but he noticed that his fingers were becoming shorter. No history of syphilis. The very clear woodcut, from a photograph, shows that the disease is not ordinary *ainhum*.

Prep. A. 102, in the Grant M. C. M., Bombay, appears to represent this condition. Bones of a foot. The metatarsal bones have been deprived of their heads by atrophy, which has also extended to the

¹ *I. M. G.* for March 1885.

shafts. The bases of the first row of the phalanges are also slightly atrophied; probably the joints here were obliterated. The shafts of the first phalanges are much atrophied, particularly the second and first. The remaining phalanges are normal. The length of the foot is proportionately diminished in front of the tarsal bones, and a general view gives the impression that the foot has been kept bound up during growth, in the manner of the Chinese.

I think that this disease, or class of diseases, well deserves further sifting and differentiation by surgeons in India. If it be neither (1) *ainhum*, *vide* page 380, nor (2) leprosy, and it is not at present at all clear that it is either of those morbid conditions, it certainly has features nearly in common with both of them. Have the patients been made cripples with a view to professional mendicancy?

In a collection of facts on the native practice of crushing in the ribs, at p. 442 of my 'Medical Jurisprudence for India,' I have shown that the great *Fragility of the Ribs of the Insane*, which has been frequently observed in Europe, occurs in native lunatics. The following appeared in a Calcutta paper:¹ 'Rib-breaking in lunatic asylums has now been repeated in all the three Presidencies. The last case is one at Madras, in which a lunatic died from having seven ribs broken on the left side, and the same number on the right.'

Rickets, a brief discussion of the question: 'How far does this condition prevail in India?' will be found at page 381.

My experience of *Strumous Disease of Bone* in India was extremely small. It may have existed in a not unhealthy-looking native girl, in whom it was evident that the bodies of two dorsal vertebræ were in process of destruction.

As I have already noticed, in the chapter on Struma, page 519, Dr. Webb and Dr. W. A. Green thought that the most common form of strumous lesion with the natives is 'bone disease of an asthenic character.' Dr. Green afterwards published² six cases in which he removed the articular cartilages of diseased joints. In one of these there was extensive malignant disease. Two cases were traumatic. The other three cases are not attributed to struma; the subjects were men of middle age.

I never saw a case of 'white swelling' of the knee originating in India. True, I saw cases of joint disease which may have been strumous, but which were not at all distinctively so, except in a young European lady with hip-disease.

Prep. 8, Ser. 3, in Sibthorpe's Madras Catalogue is 'Scrofulous ulceration of the knee-joint, taken from a native tailor whose limb was amputated above the knee. He died of phthisis shortly afterwards.'

In natives of India articular disease may generally be traced to other

¹ *Englishman*, September 26, 1871.

² *I. A.*, No. 4, p. 556.

causes than struma; such as injuries,¹ especially in ill-fed cachectic scorbutic subjects, fatty degeneration of bone, pyæmic mischief, supuration consequent upon malarial fever, or smallpox, see *Necrosis*, page 782.

The College Museum, Calcutta, has a considerable number of good specimens of joint-disease, commencing at No. 1,284, but the nationality of the subjects is not given. In the 'S. C. R.' for 1883, there was only one resection of a native elbow-joint, affected with pulpy degeneration. In Calcutta, Dr. McLeod had, in five years, 41 excisions of joints.²

The case of a sepoy with *Loose Cartilage* appears in the returns for 1880.

Prep. 9, Ser. 3, in the catalogue of the Madras collection, is loose cartilage removed from the knee-joint, by Dr. W. Johnson.

I did not see even an imported case of *Psoas Abscess* in India. In the three years ending 1882, 4 European soldiers died of this disease in all India. In these, the disease was probably imported. The returns of the native army for the three years, give 5 cases of psoas abscess, one of which was fatal. Within the same time, 4 sepoys, one of whom died, were admitted for *Lumbar Abscess*.

It will be borne in mind that psoas and lumbar abscess may occur quite independently of struma and bone-disease.

Dr. McLeod has published³ two cases of psoas abscess. One was fatal, a Hindoo woman, *æt.* 30, with a history of syphilis and salivation. There were a sinus in the left gluteal region, and an abscess in the iliac fossa, with caries of the lumbar vertebræ. In the other, a Hindoo male, *æt.* 40, there was spinal curvature with fluctuation in the lumbar and iliac regions. The collection of pus was evacuated by incision below Poupart's ligament. He left the hospital, contrary to advice, in 144 days, greatly improved in general health, and able to walk without discomfort; a small sinus, with slight serous discharge, still existing in the groin.

Dr. Wilkie, has also reported⁴ a case of psoas abscess in a native.

Parasitic Disease of bone is represented by *Chionyphe Carteri*, for which see page 45. It would appear that there are only three specimens of this remarkable disease, Nos. 143-5, at the Grant Medical College Museum, Bombay. Young officers landing there will do well

¹ As in this case,—Preparation 1123—'Parts forming the knee-joint exposed. The interior of the joint is perfectly healthy. The inner side of the condyle is superficially necrosed, and communicates with burrowing abscesses. The patient was a Mohammedan coachman, *æt.* 16. He was admitted with a lacerated wound over the inner aspect of the upper part of the right knee-joint, caused by the falling of a horse on which he was riding.' The thigh was amputated. He did not recover.

² *Operative Surgery in the Calcutta Medical College Hospital.*

³ *I. M. G.* for June 1882.

⁴ *Ibid.* for February 1878.

to examine them, as others may not occur in their experience. I saw only one case.

Several specimens are preserved in the Madras collection, *vide* pp. 135 and 150 of Sibthorpe's catalogue, 2nd edition.

DISEASES OF THE CONNECTIVE TISSUE.

Various morbid conditions prevalent in India are principally seated in the areolar tissue; among these are elephantoid hypertrophy, leprosy, guinea-worm, the œdema of malarial cachexia, and beri-beri;—for the last two see pp. 251 and 260.

Abscesses of various kinds, rarely due to struma, are very common in India. Abscess being bracketed with ulcer in the returns, it is not possible to gauge the frequency of the former condition among our *European soldiers* in India. The number of men invalided for 'abscess,' in the three years ending 1882, were 15, 27, and 15. The admissions of *European women*, during the same period, were 65, 57, and 55. The admissions and deaths of *native soldiers*, under this heading, were 2,525 with 8 deaths, 2,280 with 3 deaths, and 2,293 with 3 deaths.

When I was in charge of the dispensaries of Howrah and Ootera, opposite to Calcutta, in 1852-3, there was a great prevalence of enormous *Fascial Abscesses* of the thigh, not entirely confined to the native population.

For *Erysipelas*, *Boils*, and *Carbuncle*, see page 320, and *Diseases of the Skin*, page 796.

As would be expected from the infrequency of struma in India, Psoas and Lumbar Abscess are rare. I never saw a case in that country. See *Diseases of the Osseous System*, page 788.

Sir Joseph Fayrer has published two notices¹ of the not unfrequent occurrence of *Iliac Abscess* in natives of India as well as in Europeans. He considers that the abscesses seem 'to be referable to either fever (when, no doubt, they are the result of capillary embolism, due to the hyperinotic condition of the blood), or to such other conditions as induce suppuration in septicæmic states, to external violence, strains, or over-exertion; to sudden and perhaps inordinate muscular efforts, in which a muscular fibre, being wrenched from its attachment, tears away a fragment of periosteum, and thus induces superficial necrosis and suppuration in that area, which becomes the nucleus of the abscess.' Inability to extend the limb is a very characteristic symptom; but it varies in degree. In a European woman, *æt.* 34, upon whom Dr. Ewart operated,² there was great difficulty in walking, the

¹ *Clinical Surgery in India*, p. 602, and *Clinical and Pathological Observations*, p. 439.

² *I. A.*, No. 31, p. 160.

thigh on the affected side becoming slightly flexed, causing lameness, with inversion of the toe, and apparent shortening, but she walked from her bed to the operating table without much lameness or expression of pain. She described the sensation, in the neighbourhood of the disease, experienced in walking, as being rather an uneasiness than actual pain.

Dr. McLeod¹ reports eight cases of iliac abscess, occurring in native males and females, Europeans and East Indians. All proved successful after incision. No sinus remained. The abscess filled up in from ten to fifteen days. Cases have also been reported, in a native boy of 15 by Dr. Cayley, and in a mehtur, *æt.* 31, by Dr. W. B. Beatson.² Fayrer advocates the early opening of these abscesses, before fluctuation can be perceived. See Macpherson's case, page 694.

Obesity.—I have already mentioned that it may, I think, be accepted as a general rule that those Europeans who have long resided in India are somewhat thinner or fatter than they ought to be. The returns do not give us any useful information on this point. In the four years ending 1883, only one European soldier was invalided for obesity, and six native soldiers were admitted for treatment.

Dr. Macgregor remarked³ that neither the private European soldier nor the native is generally rendered inactive from obesity; and it is only when the former becomes a sergeant and the latter a non-commissioned officer, that unusual obesity manifests itself and renders them unfit for active duties. He adds that we often find the deposition of superabundant fat in the native soldier has the effect of producing cutaneous diseases. This arises from want of cleanliness, for the individual, as he increases in bulk, becomes lazy and careless of his person.

I have already cited (p. 19) Mr. C. Macnamara's observations⁴ on 'Fatty Degeneration of the Liver and Other Organs, considered as the chief cause of the high rate of mortality among the European troops of the H.E.I.C. in Bengal.' This paper still deserves the attention of Indian medical men. The author showed that the soldiers consumed, on an average, 76 ounces of food per diem; whereas men of the Royal Navy eat but 35 ounces daily. In Indian barracks, the men were, he contended, 'placed in the same circumstances as animals which are to be fattened.'

I removed several huge *Fatty Tumours* in natives. I have already recommended, at p. 359, attention to these cases to young civil medical officers who are anxious to make their dispensaries popular.

Foreign Substances.—One of the severest and most extensive abscesses, caused by the presence of a foreign body, which I have seen occurred in a poor European sailor, at the back of whose thigh about

¹ *I. M. G.* for June 1882.

² *I. M. G.*, vol. i. p. 60.

³ *Op. citat.*, p. 200.

⁴ Published in the *I. A.* for October 1855.

two inches of the doubly-serrated edged spinous weapon of a sting-ray had been deeply lodged for many months. Rapid healing followed its removal.

Asst.-Apothecary G. T. Carroll describes¹ the case of a native cart-driver, in the sole of whose foot a piece of glass, a little over an inch long by a quarter of an inch wide, remained for thirteen months without exciting inflammation.

A gunner came under the care of Mr. J. W. Winchester in Cutch,² having accidentally had a portion of a porcupine's quill run into the lower part of his back at the upper part of the sacrum to the left. The direction in which it had entered was 'perpendicular to the bone.' Nothing could be detected at the depth of two inches. He was discharged in a fortnight, it being doubted that the foreign body was there. He returned in a few days with severe pain and an open sinus, around which, after the good old fashion, three dozen leeches were applied! An exploratory incision having been made, and poulticing having been continued for three days, a foreign body was detected and brought out. It was the point of a porcupine's quill rather more than two inches long—consequently, its point must have been, at first, nearly five inches deep. It had been lodged nearly a month. Recovery was speedy.

Dr. McLeod has commented upon the frequency with which natives suffer from the lodgment in their flesh of *Thorns*, from the formidable spike of the *Phoenix Sylvestris* to the tiny thorns of the *Agemone Mexicana*.

When surgeon to the Chandney Native Hospital, Calcutta, where cases demanding the removal of deeply and firmly impacted foreign bodies must have occurred almost daily, my friend Dr. Herbert Baillie remarked³ that 'it sometimes happens that, a free incision having been made over the place where the object lies, it can be neither seen nor dislodged; if then a stream of water be poured over the part from some height for a short time, it will probably be found to wash out the body, or, if it fail to do so, it may loosen it and, by blanching the parts, bring it better into view, and thus allow of its being extricated.

DISEASES OF THE CUTANEOUS SYSTEM.

My remarks will be confined to the skin diseases most prevalent in India. The majority of those cutaneous affections which are common in temperate climates occur in India. Still that country has a few skin diseases of its own; and climate, food, habits of life and other circumstances so greatly modify several cutaneous maladies observed

¹ *I. M. G.* for August 1876.

² *I. M. J.*, N. S. vol. ii.

³ *I. M. G.*, vol. iv. p. 259.

in the East that these ought to be illustrated by a full treatise with an atlas of coloured plates. None could better contribute such a treatise than Dr. William J. Palmer, to whose most valuable *Rough Notes on the Common Forms of Skin Disease met with in Calcutta*,¹ I am much indebted for facts embodied in this chapter; or than Dr. A. Garden, whose learned monograph² on 'Herpes,' 'Disorders of Pigmentation,' and 'Phytodermata, Parasitic Skin-diseases,' might be made an excellent nucleus for a standard work.

Of *Erysipelas* as it occurs in India, I have given an account at page 320. I do not think that a clear line of distinction can always be drawn between *Erythema* and *erysipelas*; but we saw a great deal of what was termed 'erythema' when *erysipelas* became prevalent in Calcutta in the year 1873.

One of my brother officers in Calcutta, had an attack of *Erythema nodosum*.

Roseola and *Urticaria* give place to *Lichen tropicus* in India. There, we always use ginger-powder frictions in cholera. In fair Europeans who recover from this disease, there is sometimes an eruption, much like *roseola*, over the whole body.

I am not aware that true *Pellagra*, established as a chronic disease, with grave constitutional symptoms, exists in India among either natives or Europeans. In the East the latter do not work in the sun as do the peasants of those southern countries of Europe in which *pellagra* prevails; but, in India, violent *Sunburn* occurs, especially in very fair Europeans. A young Madras officer left Chittagong with his wife and infant. The native boat on which they embarked was swamped and he only survived after floating about, clinging to part of the wreck, for nearly twenty-four hours. When he was brought into the station, the back of his neck and the upper part of his shoulders which, although constantly wet, were generally above water, were one terrible sore, like a neglected scald. I had in my ward a healthy, red-headed European, whose back the sun had struck while he was wet after bathing. A pint of boiling water could hardly have caused severer vesication. This danger is well known to experienced European soldiers.

I saw several very severe cases of *Eczema*, in all its forms, in natives and others in Calcutta.

Assistant-Surgeon O. C. Chatterjee has very strongly recommended³ an ointment of animal (bone) charcoal as 'a specific in *eczema*.'

Intertrigo of course occurs. Chafing is doubtless one of the causes which determine the chronic inveteracy of those skin diseases which, in India, so frequently affect the axillæ and insides of the thigh.

I had a case of *Impetigo* of the legs in an old nurse, who, I believe, brought it from England, but not in any other case.

¹ Re-published from the *I. M. G.*, 1873.

² Published in the *I. M. G.* for 1874 and 1875. ³ *I. M. G.* for 1882, p. 96.

I never saw a case of well-developed *Rupia prominens* in India. *Ecthyma* is occasionally seen there in cases of tertiary syphilis.

In the report on Cannanore, Malabar Coast (Madras Topographical Report, p. 11), it is stated that cutaneous eruptions are very prevalent, and that scabies (Malabar Itch) is thought by some to be more common and more difficult of cure than in most other parts of India. This, however, is not found to be the case, but an eruption *Ecthyma cachectica*, often mistaken for scabies, is very difficult to cure. It is attributed by the natives to living almost exclusively on fish, is occasionally attended with anasarca of the lower extremities, and is liable to be mistaken for scabies purulenta, from which, however, it is distinguished by the absence of itching. Then follows the odd remark that 'The attempt to cure it by sulphur has been observed, in some instances, to occasion general anasarca, followed sometimes by inflammation of the lungs.' See Scabies, page 805.

Prurigo occurs nearly as in Europe.

Lichen tropicus. Prickly heat especially annoys those of fair complexion recently arrived in India; but few Europeans, resident in Lower Bengal, wholly escape it in any hot season. I often had a full crop, but the eruption caused so little distress that I generally told complainants that the rash was salutary and ought to be appreciated with satisfaction. I said this for the last time to a sailor in my ward as, very shortly afterwards, the season being an unusually hot one, I suffered so severely in my own person, that I never afterwards regarded prickly heat as a subject for levity. I was very feverish and my banyan stuck to my back. I have already alluded, p. 13, to the case of a young English lady whose death, about three weeks after her first confinement, appeared to be determined by the irritation of prickly heat.

Psoriasis vulgaris (*Lepra vulgaris*) did not originate in India, in any European, within my observation. Dr. Coates¹ cured aggravated psoriasis by glacial acetic acid.

I saw *Psoriasis palmaris*, but never *P. labialis*.

Miliaria occur, their contents being either clear or puriform. Dr. R. D. Murray has given in full² the cases of three young native firemen, admitted in August and September, 1885, and February 1886, to the Calcutta General Hospital, from three different steamships with *Acute Miliaria* or *Miliary Fever*. An abstract of the first case gives the typical character of the malady: Was admitted with fever of three days' and an eruption of one day's duration. Face and entire body covered with a profuse, irregularly scattered eruption of small clear vesicles, very like sudamina, of the size of millet-seeds; discrete on the trunk and limbs, but confluent on the face, neck, and ears. A few large umbilicated vesicles on the front of the chest. 'Appearance of the

¹ I. M. G. for 1883.

I. M. G. for March 1886.

face very much as in measles or confluent smallpox; it is red and swollen (*miliaria rubra*) eyes half-closed and suffused, lacrymation, running at the nose, sneezing, and all the symptoms of smart coryza. A sour, unpleasant odour from the body, also suggestive of smallpox. Face deeply pock-marked. Temperature 101° , pulse 85.' In a few days, he was discharged quite well. Pyrexia and other symptoms rapidly subsided; the eruption gradually became milky (*Miliaria alba*) and then dried up and fell off as branny scales. The entire cuticle of the face and ears peeled off.

Dr. Garden's monograph on *Herpes*, cited at the head of this chapter, forms a library of reference on the subject of this very remarkable group of maladies, as occurring both in India and in Europe.

I had more than one case of severe intercostal neuralgia with *Herpes zoster pectoralis* in natives. Dr. Garden found that, in 4,493 cases of skin disease treated in the Saharunpore (native) dispensary in five years, 116, or 2.6 per cent., were cases of herpes. Of these, 85, or 1.28 per cent., of the total number of skin-diseases, were cases of zoster. Among the small European and Eurasian community of that station not exceeding 300, Dr. Garden saw only three cases—two females and one male in eight years. Meeting, in the spring of 1884, a distinguished brother-officer lately retired, he complained of intercostal pain in the right side. Herpes zoster was discussed and he agreed to apply a sinapism. He went through a marked attack of Herpes zoster. He told me that he had heard of two other retired Indian officers who had been thus attacked not very long after settling at home.

There is a report, by Dr. G. H. Savage, on *Herpetic Fever* as it prevailed in London. This notice appeared in the 'Lancet' on January 20, 1883. On the first of that month, a member of my family had a large spot of herpes on the upper lip. This recurred on the 27th. On the first, I heard that one of the maidservants was suffering much from pain in the neck. I found several lymphatic glands, on one side posteriorly, much enlarged and very tender. Just as I was about to apply iodine paint, I turned up the hair and found a spot of herpes, nearly as large as a florin, just above the upper gland.

It is mentioned, in a Report on the Diseases of Singapore,¹ that herpetic eruptions are very common there, and that European remedies are employed in vain; but that, by rubbing the leaves of the *Cassia latifolia* on the affected parts an almost magical effect is produced in many instances. The application of pure sandal-wood oil is also very effectual. Here the designation 'Herpes' appears to be applied in error. We are told² that 'Herpes' (particularly *H. circinatus*, is prevalent on the Malabar coast. Of *H. circinatus*, 15 cases were treated at the Saharunpore Dispensary. In the majority of cases, the duration

¹ Madras Quarterly Medical Journal, vol. i. p. 74.

² Madras Topographical Reports, Cannanore, p. 11.

was a month. The number of cases of *H. iris* was 12. For *Herpes ophthalmicus*, see *Diseases of the Eye*. Page 458.

I attended the infant of a German professor married to an East Indian. It was born puny and, being attacked with *Acute Pemphigus* was so almost absolutely covered with blebs that it sank under the irritation, nearly as if it had been scalded to death.

Dr. Palmer saw five cases among his dispensary patients.

In 1881, 3 cases of pemphigus occurred in the 6,548 children of European corps in India.

Dr. Stewart published¹ a case of this by no means common skin disease in a young Englishwoman recently arrived in Calcutta, and nursing an infant. The bullæ were of all sizes, from that of a pea to that of an egg. The constitutional disturbance was very moderate. She recovered in about a fortnight.

Dr. Lyons² cited an old case of this eruption which he found reported in the case books of the Calcutta General Hospital in 1823, and a similar one which came under his treatment there in 1871.

Acne occurs in India, but not so generally as in England, where cutaneous action is less free and is liable to frequent check. In a highly valuable paper on scurvy, mainly founded upon his experience of the disease in the Afghan War of 1878–81 and in the Nagpore Jail from November 1881, my friend Dr. R. Temple Wright notices³ that ‘At the beginning of the cold weather of 1884, it was noticed that the prisoners who had scurvy most severely, were also affected with acne on the face. The same thing was observed in 1883, when the coincidence was thought to be accidental; but, as it occurred again in 1884, must we consider that scurvy *plus* acne indicates a worse cachexia than scurvy alone? In 1885, most of these prisoners had quite lost scurvy, but acne was still present, though to a less extent.’⁴ I saw very obstinate cases of *A. indurata* in a young married English lady, and in a youth of mixed blood. A middle-aged lady was much disfigured by *Acne rosacea*, but I believe enjoyed good health, and survived her return to England many years.

I once saw *Sycosis* of the chin in a native—not my own patient. Dr. Palmer also met with only one case of *sycosis menti* in his search for skin diseases. In this case, the inflammation was certainly outside and around the hair-follicles; for, when a hair was pulled out, the lining membrane of the follicle came with it, covered with a thin layer of puriform matter; no such alteration being found in the hair itself

¹ *I. M. J.*, vol. i. N. S. p. 117

² *I. M. G.*, May 1, 1871.

³ *I. M. G.* for February and March 1886.

⁴ Did the designation ‘Poor Jack Nasty-face,’ commonly applied to the British seaman of last century—who was, doubtless, rarely free from scorbutus—and the common vernacular expression ‘scorbutic eruption,’ take their origin from this circumstance?

as is seen in *tinea tonsurans* or other cryptogamic diseases affecting hairy parts of the body.

I was shown a case in which very well-marked *Ichthyosis* affected the legs of a native.

I have spoken of *Leucoderma* in the chapter on Leprosy, page 376.

Albinos are occasionally seen among the natives. Mr. C. Macnamara mentions that they are regarded, in Bengal, as lepers and are not permitted to marry. This rule cannot be absolute, as there used to be an albino family in Dacca. The Albinism of India is as complete as that of Europe.

My friend Dr. Francis recorded and figured¹ the case of a Russian Finn seaman, *æt.* 43, the subject of *Melanopathia* or *Melasma*. The coloured patches were inclined to be symmetrical. There were deposits of buff-coloured pigment in irregular patches on the face, neck, trunk, and extremities. They appeared to be in the rete-mucosum, the epidermis seeming to be quite unaffected. The change began about eight years previously, when he first visited the tropics. It first appeared near the umbilicus, spreading thence. He had always been in perfect health and was a stout, strong man. The piebald, full-length figure which illustrates the report is a rude work of art, but maps out the large discoloured surfaces correctly. I have the original photograph.

An esteemed brother officer appeared to suffer from *Melasma Addisoni*. (*Vide Diseases of the Renal Capsules*, p. 666.) Unless they are in the habit of going about in the sun—which they very rarely are—Europeans are far more exempt from *Freckles* in India than they are at home. I do not think that I ever saw a freckle in a European lady's child.

A few cases of *Frostbite* and *Chilblain* appear in the returns, having doubtless occurred in service far north and in soldiers' children at stations in Upper India where the winter cold is rather severe.

Furunculi are among the severest minor diseases of Europeans in India. There, as in England, they may occur at any time of the year, but are most prevalent during the six weeks of the very hottest season which precedes the setting-in of the rains, about June 20. They do not visit us every year. Thus I had severe boils once, in cool weather, before I went to India, and in three hot seasons in India. In certain years, boils are distinctly epidemic. In one year, at Howrah, nearly every European child had its face closely spotted with unusually small (about the size of a pea) livid boils, which gave out, not pus, but a sanguinolent serum. There did not appear to be any constitutional distress in most of these cases; but, in England and once in India, I felt unspeakably 'low' when the subject of boils. I do not doubt the correctness of the theory that boils are immediately caused by the plugging of over-taxed cutaneous vessels by blood-pigment.

¹ *I. M. G.*, vol. iv. p. 15.

I have known infants at the breast to be in great danger from boils, especially on the occiput. In adults, the prophylactics and remedies are plenty of 'support' and quinine. A chaplain—a very moderate liver—confirmed my experience, obtained long previously in my own person, when he said, 'I always double my allowance of beer when I have boils.' In my remarks on puncture in liver-abscess and on Dr. Pancoast's 'Therapeutic Knife,' I have mentioned that I found very early puncture most useful in rendering boils abortive.

That excessive stress upon the sudiparous function of the skin, in extremely hot weather, is the immediate cause of boils is probably the opinion of every medical man who has practised in India, and the fact is especially illustrated in the narrative of John Zephaniah Holwell, the surgeon who was twice Governor of Bengal;¹ but this does not explain the occurrence of boils in English cool weather.

In India, poulticing often brings out a crop of boils around the edges where the air and the poultice meet.

At a meeting of the Epidemiological Society, in November 1884, it was mentioned by Inspector-General Lawson and Surgeon-General Gordon that they believe that boils are not prevalent, or do not occur, in the West Indies or on the West Coast of Africa.

See remarks on the *concurrence of Boils with Hepatic Abscess*—page 622, and with *Diabetes*, p. 372.

I regard boils as a constitutional disease of which the cutaneous disorder is merely an expression.

The statistics of boils for the four years ending 1883 are interesting as showing their great prevalence and severity (as no trivial case of boils would be entered in the returns) among European and native soldiers and prisoners. It will be noticed that, in 1880, a European child, a native soldier, and a prisoner appear to have died under this cachexia. European women 12, 15, 12, 11—Children, 59, one death, 36, 52, 50. European soldiers, 1,057; 1,400; 1,168; 1,401. Native soldiers, 4,396, one death; 4,385; 4,970, 3,923. Prisoners 1,449, one death; 1,303; 1,162; 1,413.

We often meet with cases in which it is very difficult to draw a clear line between boil and carbuncle. A well-to-do East Indian had a very localised carbuncle on his thigh. I made a crucial incision through a slough which had much the look and size of a rotten chestnut. He recovered precisely as he would have done from a common boil.

Carbuncle has always interested me. One of my earliest publica-

¹ Holwell says that, on being released from the Black Hole, he 'became covered from head to foot with large painful boils, the first symptoms of my recovery; for, until these appeared, my fever did not leave me.' Again, 'Here the other gentlemen broke out likewise in boils all over their bodies (a happy circumstance which, as I afterwards learned, attended everyone who came out of the Black Hole).'

tions was on 'The Occurrence of Inflammation of Serous and other Tissues in Cases of Anthrax' [*hominis*].¹ Carbuncle is rather frequent among elderly and rich natives, especially in the diabetic. A large proportion of these cases are fatal. I have noticed, under the heading of Diabetes, page 372 (note) that Baboo K. Acherjee was led to discover diabetes in two well-to-do natives, by the characters of carbuncles and boils from which they suffered. A brother officer was much overworked at a time when he had dropped all stimulants, having been jaundiced. His first symptom of breaking down was a fainting fit; he then got carbuncle, and was obliged to take sick leave to England. A friend of mine, a healthy indigo-planter, advanced in life, came home, after very protracted residence in a healthy Indian station. He got smallpox in a London lodging, and recovered in the hospital for that disease. On his discharge he got a carbuncle at the back of his neck, which killed him. I know of two other cases, in retired Indian officers, who recovered. I have repeatedly seen cases in which I considered that anthrax was a form of senile gangrene. In the London cases of carbuncle which I published in 1841, the fatal complications were evidently caused by *ichoræmia*. In India, a case was reported² in which Mr. — aged 36, who had been fifteen years in the country, living a very sedentary life, and being almost a total vegetarian and teetotaler, except on rare occasions, when he took animal food and wine freely, was attacked with a carbuncle at the back of the neck. He sank on the fourteenth day, some hours after imprudently taking a cold bath. The lungs were studded with secondary deposits of pus varying in size from a pin's head to that of a split pea. There was a long and firm blood-clot in the left vena innominata, but no pus could be detected in it, nor was there any sign of phlebitis.

The returns for the four years ending 1883, give only one admission of a European woman; no case in a child; European soldiers, 13, 8, 10, 7 admissions; native soldiers, 29, 41, 34, and 26 admissions—four fatal cases in the first three years; prisoners (among whom many are advanced in life) 138, 145, 131 and 119. In the first three years five fatal cases occurred in jails.

A European officer, of great literary ability, had all his finger-nails deformed by *Chronic Onychia*, probably of gouty origin.

In-growth of the Toe-nail.—I have seen many cases in Europeans. In an unfortunate man who was supposed to be the Nana, to examine whom the Government sent me up to Cawnpore, I looked in vain for the very slightest trace of this affection, from which I was told the real miscreant had suffered.

I found repeatedly that, when the disease is taken early enough, careful cutting of the ingrowing nail and the use of easy boots are

¹ *The London Medical Gazette* for 1841, pp. 82 and 105.

² *I. A.*, No. 3, p. 463.

quite effectual. In rather more advanced cases, we check the evil by keeping a strip of lint or thin white kid between the nail and the soft parts. Dr. R. C. Sanders does not remove the in-growing nail. It grows again and is just as bad as its predecessor; but he 'well removes the flesh into which the nail grows.'

Dr. John Shortt¹ never met with a case of in-growing of the toe-nail in a shoeless or loose-slipper-wearing native. Observing that the nail naturally has a more or less convex form, he considers that our object, in the first instance, is to reverse this natural form of the nail by rendering its convexity concave by shaving, with a sharp pen-knife, the centre of the nail, to the breadth of a line, from its root to its apex, this part being so thinned as 'almost to make the blood start through.' Small pellets of cotton-wool, well dusted with powdered burnt alum, should then be gently inserted, with the aid of a probe, under either end [side] of the nail, and, every four or five days, the shaving should be repeated, the pellets being renewed and slightly enlarged on each occasion. He had tested this plan for fifteen years, in a large number of cases with perfect success, cure being effected in ten days or a fortnight.

Whitlow occurs, but far less commonly than at home. Europeans who live by handicrafts are not numerous, and most natives keep their hands scrupulously clean and their nails in good order.

Natives who do not wear shoes are free from *Corns*, and few well-to-do Europeans are of sufficiently pedestrian habits to get them. In 1881, only fifteen admissions for 'corns and bunions' appear in the returns of the whole native army.

Cornua. Prep. 249 in the C. M. C. M. is described as a horny growth, about four inches in length, half an inch thick at the apex, and an inch thick at the base, and slightly curved at the central part, removed by Professor Allan Webb from the back of the thigh of a patient in the native hospital. Another case is reported in the Bombay Med. and Phys. Trans., vol. ii., for 1839.

Mr. J. C. Wilson² narrates the case of a Hindoo man, *æt.* 22, in whom a horn-like excrescence had been growing from the top of his head for five years. It, at first, grew straight, round, and pointed to the length of five inches, when it was cut off, through its substance, by a barber. This had been done three or four times. The base was surrounded by elevated integument. It was ribbed longitudinally, was waxy in colour, and hard, and possessed all the external appearance of horn.

Mr. Samuel P. Johns reported³ the case of a Hindoo, *æt.* 45, in whom a horn-like body had been growing for sixteen months from the right side of the abdomen. It was $3\frac{1}{2}$ inches long, and four inches in

¹ *I. M. G.*, vol. i. p. 255.

² *Ibid.*, vol. i. p. 152.

³ *Ibid.*, p. 337.

circumference at its root. Like a horn, it was round and pointed, slightly curved at its end, and surrounded by hard and elevated skin at its root. It had no connection whatever with the structures beneath. It was preserved in the Museum of the Agra Medical School.

Dr. Kenneth McLeod¹ removed a horn-like conical mass, 2 inches long and $2\frac{1}{8}$ inches in diameter at base, which had commenced about a year previously as a small wart-like growth on the right of the sternum of a Hindoo man, *æt.* 35. It was of the colour of light horn, and was apparently composed of an agglomeration of vertical columns, striated externally. The base was exquisitely tender and was somewhat bulbous. The surrounding skin was healthy. It was easily removed, and he recovered without a bad symptom. It was sent to the C. M. C. Museum. Dr. Colles stripped down one or two of the columns of which the growth consisted, and found that it was a purely epithelial overgrowth.

Dr. K. McLeod has reported² the case of a Hindoo, *æt.* 60, in whom circumcision had been performed eight months previously for phimosis with preputial irritation. 'There were two well-marked horny projections, growing from the corona glandis, and the surface of the glans was covered with a cap of similar material.' Amputation of the glans, including the growth, was performed with satisfactory result.

Leprosy, *vide* page 375.

Lupus and Keloid, *vide* Tumours, page 357.

Molluscum contagiosum is very rare in Bengal Proper. It did not occur among Dr. Palmer's reported dispensary cases. Baboo O. C. Dey speaks³ of it, in rather obscure terms, as if it were of common occurrence at Cawnpore.

Visiting at the house of a wealthy Calcutta Hindoo, I saw a child between two and three years old, which I perceived, at a glance, was the subject of this disease. The fact that other children in the house were affected proved that the malady was contagious.

Tumor adiposus. See Diseases of the Connective Tissue, p. 790.

Nævus vasculosus. Prep. 242 in Ewart's Catalogue is a section of a vascular tumour, as large as a walnut, removed by Sir Joseph Fayrer from the upper and outer part of the arm of a native of Jessore. It consists of a rich network of vessels supported by fine delicate fibro-areolar tissue. Prep. 243 is a section of a pendulous fibro-vascular tumour, removed from the anterior superior spinous process of the ilium of a native woman of 60. It consists of an extremely fine network of areolar tissue and minute vessels. Prep. 244 is an aneurismal tumour by anastomosis, occupying the situation of the middle and

¹ *I. M. G.* for 1868, p. 63.

² *Ibid.* for January 1886, p. 27.

³ *Ibid.*, vol. ii. p. 8.

ring fingers. The arteries and superficial veins are enormously enlarged.

Under Injuries to the Skull, I have spoken, p. 394, of the fractured cranium of a little Cookie girl, the calvarial bones of which were enormously thickened, having formed part of huge vascular tumours. Dr. Esdaile recorded and figured¹ the case of a Hindoo prisoner, *æt.* 30, in whom there was a large pulsating swelling which rendered the crown of the head conical. All the arteries of the left side of the head were greatly enlarged and beat strongly. Immediately on tying the common carotid artery on that side, the swelling relaxed, and all pulsation ceased. Fourteen days later the ligature had separated, the swelling was daily decreasing, the integuments were quite lax and a radical cure was anticipated.

My friend Mr. Partridge reported² (1) the case of an Eurasian girl, *æt.* 18, in whom a congenital venous tumour occupied very extensively one side of the face extending slightly into the orbit, everting the lower lip, and involving half of the tongue. The injection of about an ounce of a saturated watery solution of tannin at several different points, at once produced complete consolidation and great distension of the tumour. The constitutional disturbance which ensued was trifling, and the tumour began perceptibly to decrease in size. There was some sloughing of the lower lip, but the operation promised to be essentially successful when she was fatally attacked by cholera.

(2) A Hindoo woman, *æt.* 36, had an arterial tumour or aneurism by anastomosis, probably congenital, which gave a most extraordinary nodulated appearance to the whole of the right auricle, and extended over nearly the whole of the temporal region and behind the ear nearly as far as the occiput. It pulsated strongly, and appeared to involve the branches of the temporal and also the posterior auricular and occipital arteries. The auricular part was somewhat ulcerated, and a somewhat serious burst of hæmorrhage had occurred from this surface. The treatment employed in the first case was adopted. There was considerable constitutional disturbance. On the second day, there was rather serious hæmorrhage from a part of the tumour, where the growth was still soft and pulsating. The consolidated mass appeared to undergo a gradual process of absorption, and it was only in the auricle, where the covering integument had originally been excessively attenuated, that a little separation by sloughing took place.

I saw a very small *Port-wine Stain* in a European girl born in India.

Elephantiasis Arabum is considered under the head of Malarial Cachexia, p. 262. *True Leprosy*, at p. 375.

As there are few Europeans in India between the ages of 7 and 17,

¹ *I. M. J.*, vol. ii. (Finch's) p. 193.

² *I. M. G.* vol. i. p. 36.

Warts are rarer there than at home, but some Europeans are much troubled by obstinate verrucæ. They are not common in natives.

Condylomata occur as in Europe, as also do *Sebaceous Tumours*.

Molluscum fibrosum, see *Tumours*, p. 356.

Yaws and Delhi Boil. That eminent and venerable medical philosopher, the late Dr. Gavin Milroy, has fully dealt with the subject of *Frambœsia*. Sir Joseph Fayrer¹ and Dr. John Murray² have described *Delhi boil*. In his article on *Yaws*, in 'Quain,' Sir Erasmus Wilson remarked that the malady 'is peculiar to the African race, both in their native country and in the West Indies.' But the Cingalese, who are not negroes, suffer greatly from this disease.

It is stated, in the *Madras Topographical Reports*,³ that '*Frambœsia*' or the *Yaws* is not uncommon at Moulmein among the native population, and is called by them 'tongo aua.' It is classed with venereal affections. The remedy chiefly relied on for its cure is the *Smilax China*, of which O'Shaughnessy Brooke says that it is much, and he believes, advantageously used by the native practitioners of Calcutta, as a substitute for sarsaparilla. Waring, however, says that, as found in Indian bazaars, it is usually worm-eaten and worthless. I have not seen any other notice of *yaws* as prevailing in the Tenasserim Provinces.

Dr. Milroy showed⁴ that he had gathered from the reports of Drs. Loos and Danforth and from Marshall's '*Medical Topography of Ceylon*,' that a form of *Parangi disease*, as it occurs in that country, closely resembles, in many respects, *yaws* in its bad and chronic form. The drawings which illustrated the reports of Dr. Kinsey on *parangi disease*, are deposited in the library of the College of Physicians. I believe that Dr. Milroy and Dr. Murray were agreed that they display both *yaws* and *Delhi boil*. This inquiry well deserves the fullest attention. I saw *Delhi boil* at Delhi and in the Calcutta Medical College Hospital. In passing through a native crowd in the bazaar of Old Cairo, I saw a man (probably an Arab) with an indisputable *Delhi boil* on his face.

Surgeon-General J. Murray considers that *Delhi boil* is due to a parasite (undetected) in *Delhi water*. On this point see articles by Dr. J. Fleming.⁵

A *résumé* of Dr. D. D. Cunningham's recent investigations into the nature of *Delhi boil* is cited in the '*I. M. G.*' for February 1866, from the Sanitary Commissioner's Report. This eminent authority describes the lesion as a simple granuloma associated with and very possibly due

¹ Quain's Dictionary.

² *Transactions of the Epidemiological Society of London*, vol. ii. 1882-83.

³ *Tenasserim Provinces* p. 176.

⁴ *M. T.*, November, 1878, p. 516.

⁵ *I. M. G.*, vol. iv. p. 233, and Staff-Surg.-Major A. Smith, *Army Medical Department Report* for 1863.

to the presence of a peculiar form of parasitic organism. No opportunities were afforded of obtaining an insight into the developmental history of these bodies, but Dr. Cunningham is inclined to regard them, provisionally, as representing various stages of some simple organism of mycetozoic origin—probably parent plasmodia or amœbæ.

Pruritus occurs as in Europe. I had some troublesome cases.

Hyperidrosis.—Some very marked cases have been observed in natives, especially one recorded by Mr. A. Duncan,¹ in which a native recruit, a stout, able-bodied, healthy-looking man in whom, on the slightest exertion, moisture oozed profusely from his hands and feet. The word '*attention*' being given, forthwith, on clasping his hands, and that not very forcibly, the perspiration poured, rather than dropped from them, the ground was also bedewed around the edges of his feet, and on his changing his place, there were his foot-prints in thorough wet. Being desired to step about on a cow-dung floor, every step was imprinted wet. His body remained unaffected. He stated that he had been *all his life* subject to this affection, that he was able to work like his neighbours, but got fatigued rather soon. As far as he knew, the various excretions were the same in quantity and kind as those of others. Indeed it appeared that, in all other respects, he was healthy; but he was evidently unfit for military service, as the grasping of a musket brought clear sweat streaming from his hands.

Sub-Assist.-Surgeon K. N. Acherjee slightly reported² the case of a native man, of Chuckdigee, Lower Bengal, *æt.* 26, who had suffered '*for a year*' from continual sweating of only his hands and feet. The flow was continuous and the same in quantity all day and night, and during all seasons. 'The drainage of fluid was so copious, that nearly a pint might be collected in twenty-four hours. His previous and present history spoke of no ill-health.' See *Burning of the Feet*, p. 260.

In the moist climate of Lower Bengal one does not so much suffer from being hot as from being wet. I used to watch the young officers at the Government House balls on the Queen's birthday. Most of them evidently became thoroughly wet through. An elderly medical officer, who had not perspired inordinately during his early Bengal career, found, latterly, that, on his return from his morning's work, his shirt was absolutely saturated, and, as he said, '*sloshed*' when he threw it on the floor. He took this as a warning to retire from the service. This annoyance remained in abeyance during his first eight years' residence in England, and then showed a tendency to recur upon slight exertion. The memoir of the Rev. Dr. Duff contains frequent allusions to his suffering from this malarial neurosis, which appears to have affected him more in America and Scotland than in Bengal.

Tinea tonsdens.—Dr. Palmer observes that this condition is by no

¹ *Transactions of the Medical and Physical Society of Bombay*, vol. i.

² *I. M. G.*, vol. i. p. 218.

means uncommon in the natives of Lower Bengal. A little more than five in the thousand of the healthy emigrants examined being affected.

A malady which causes a great deal of suffering in Lower Bengal, both among Europeans and natives, is a form of *Tinea*,¹ called by the natives *Dād*, and by Europeans 'Dhobie's itch.' It is clear that this is the affection which has been called 'Burmese itch.'²

Dr. Palmer says that it is so common among Europeans in Calcutta, that it constituted nearly half of the number of skin diseases seen in them in two years.

The filthy habit which natives have of not taking off the cotton *dhotee* (waist-cloth) when they bathe in the river, and of allowing this garment to dry upon them, in all probability frequently determines the occurrence of this eruption all over that portion of their bodies which very short drawers would cover. Some Europeans suffer extremely, the parts attacked being principally the upper and inner surfaces of the thighs, scrotum, and armpits. Early in my career, I failed entirely to cure, using sulphur and mercurial and ioduretted applications, but I repeatedly found that, after resisting treatment for months, the eruption would suddenly disappear on a change of season, as from hot to cold or the reverse. I am much indebted to my friend, Dr. William Palmer, for the following judicious observations upon this disease. 'It can be cured by any of the parasiticides, even by borax, but they must be applied intelligently, by persons who know anatomy, &c. Our old friend — used to doubt its curability, and he was neither careless nor captious. How are such diverse opinions to be reconciled? If the affection of the skin depends upon a *spore* germinating within it, then *every* single spore, however deep it may have imbedded itself, must be destroyed before the disease can be eradicated. The fact that the true skin (not merely the scarf skin, as in *T. versicolor*) is involved in this affection, indicates that the germs occupy the deeper layers of the dermis, as well as the surface ones; but no sooner are the multitude of germs in the surface layers destroyed, than complete relief is felt, the remedies are discontinued, and, after a month or two, a few germs in the deeper layers will have multiplied, and the disease be again as bad as ever. The sanguine doctor who promised a cure will be called hard names, the patient will lose faith in the remedy,

¹ This disease is undoubtedly a form of *tinea* or ringworm, but I have witnessed a great deal of confusion arising from calling it by other names. Thus, I was told of the sufferings of a deceased Bengal officer of my acquaintance from 'eczema.' Not readily accepting this view of the malady, I heard more, and felt convinced that the case was one of 'Dhobie's itch.' Certain excellent authorities speak of the efficacy of Goa-powder in 'obstinate psoriasis,' which one of them considers to be a form of malarial cachexia. I think that this again was Dhobie's itch.

² *Lancet*, August 2, p. 185, and August 30, p. 335, 1879.

and, after years, the skin will have assumed that thickened, dense character, where some skill, with an unusual amount of patience and perseverance, will be required to bring about a cure. I feel very certain, however, that a cure may be effected whenever the remedy is used with knowledge and faith.'

Dr. Kenneth McLeod¹ has used acetic acid with success in very severe cases of this disease. At Sir Joseph Fayrer's suggestion, I used Goa powder effectually. I had some experience of the good effects of a lotion prescribed by Dr. Joseph Ewart, of Brighton. The formula is,

R

Hydrarg. Bichlorid. gr. x.

Spir. Terebinth. ℥ss.

— Rectificat. ℥ijss. *Misce.*

A portion is brushed over the parts affected every night at bed-time. The first few applications cause considerable smarting and stinging, which soon pass off. Subsequently, little inconvenience is occasioned. Dr. Ewart writes, 'It completely relieves the itching which, in the hot weather and rains, is so troublesome as to lead to much disturbed sleep and great irritation in the day-time, especially during even moderate exercise.' 'But,' he adds, 'at these seasons it is necessary to keep up the daily use of the liniment often for weeks after the disease is apparently cured.'

Dr. Palmer observed that, in Calcutta, *Favus* is as uncommon as in London; only two cases of it having been seen in two years.

Tinea versicolor occurs in natives and Europeans. In the latter it is rarely complained of. In the former, it is viewed with that suspicion (of leprosy) with which all chronic discoloration of the skin is regarded.

Scabies is very prevalent among natives in India. I have mentioned, in speaking of Malarial Remittent Fever, page 201, that, in many patients suffering from fever and other diseases, the skin is so universally occupied by this malady, that one of the first measures needful is to cure the itch, which seldom long resists sulphur wash. Dr. Palmer says that, of 491 cases of skin disease seen at his dispensary, 488 were the victims of *acarus scabiei*. The native servants so frequently have itch that ladies and children often suffer. I always found a few applications of a weak solution of corrosive sublimate in rose water effectual. At Chittagong, I and one of my children suffered from a form of *purulent itch*. I have never been on the Malabar coast, but I believe that the disease with which we and others were visited at that seaside place was true *Malabar Itch*. I did not allow it to extend beyond the hands. All along the insides of the fingers, large, oval bleb-like

¹ *I. M. G.* for 1883, p. 198.

yellow pustules formed, causing a good deal of irritation and pain ; these breaking and discharging serous pus, there remained a thin white slough of the appearance and thickness of tissue paper. This, when seized with forceps, readily came away. Sulphur ointment speedily cured us. It will be seen, in the remarks on *Ecthyma*, p. 793, that it has been considered that ' Malabar Itch ' is not purulent scabies but ' *ecthyma cachectica*.' I am not qualified to speak with certainty of Malabar itch, but I believed and still think that we suffered from it. Our eruption was very acute and short-lived, never went on to form scabs, yielded speedily to sulphur, and was distinctly contagious ; in fact it did not resemble in any way *ecthyma* as I have seen that skin disease.

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